THE LIBRARY OF CONGRESS
1800

Class RM101

Book F7

Copyright No. 1924

COPYRIGHT DEPOSIT.

THE GEORGE BLUMER
EDITION OF
BILLINGS-FORCHHEIMER'S
THERAPEUSIS OF INTERNAL DISEASES

VOLUME V

THE GEORGE BLUMER
EDITION OF
BILLINGS-FORCHHEIMER'S

THERAPEUSIS OF INTERNAL DISEASES

CARE AND MANAGEMENT OF MALADIES
AND AILMENTS OTHER THAN SURGICAL

Forchheimer, Frederick.

VOLUME V

D. APPLETON AND COMPANY
NEW YORK 1924 LONDON

RM101
F7
1924.

COPYRIGHT, 1913, 1914, 1917, 1924, BY
D. APPLETON AND COMPANY

PRINTED IN THE UNITED STATES OF AMERICA

NOV 12 '24
©C1A808767

no 1

EDITED BY

GEORGE BLUMER, M.A. (YALE), M.D.

DAVID P. SMITH CLINICAL PROFESSOR OF MEDICINE, YALE UNIVERSITY SCHOOL OF MEDICINE,
ATTENDING PHYSICIAN TO THE NEW HAVEN HOSPITAL

FORMER EDITORS

FREDERICK FORCHHEIMER, Sc.D. (HARV.), M.D.

FORMERLY PROFESSOR OF MEDICINE, MEDICAL DEPARTMENT, UNIVERSITY OF CINCINNATI
(OHIO-MIAMI MEDICAL COLLEGE), PHYSICIAN TO CINCINNATI HOSPITAL

FRANK BILLINGS, Sc.D. (HARV.), M.D.

PROFESSOR OF MEDICINE, UNIVERSITY OF CHICAGO AND RUSH MEDICAL COLLEGE, CHICAGO

ERNEST E. IRONS, PH.D., M.D.

CLINICAL PROFESSOR OF MEDICINE, RUSH MEDICAL COLLEGE, CHICAGO

CONTRIBUTORS TO VOLUME V

MAUDE E. ABBOTT, B.A., M.D., L.R.C.P. and S. (Edin., Glasg.)
Assistant Professor of Medical Research, McGill University, Montreal, Professor of Pathology and Bacteriology, Woman's Medical College of Pennsylvania, Philadelphia

Congenital Heart Disease

GEORGE BLUMER, A.B., M.D.
David P. Smith Clinical Professor of Medicine, Yale University School of Medicine, Attending Physician to the New Haven Hospital

Diseases of the Lungs, Diseases of the Mediastinum, Diseases of the Muscles, Mechanical and Circulatory Lesions of the Kidneys

JOHN W. BRADY, A.B., M.D.
Instructor in Industrial Medicine, Harvard School of Public Health, Member on Staff of Massachusetts General Hospital

Diseases Due to Physical Agencies

HARLOW BROOKS, M.D.
Professor of Clinical Medicine, New York University and Bellevue Hospital Medical College, Visiting Physician to the City Hospital, Consulting Physician to the Beth Israel, Montefiore and Union Hospitals, and to the Hospital for Joint Diseases, New York City

Thrombosis, Embolism and Phlebitis

JOSEPH A. CAPPS, A.M., M.D.
Professor of Medicine, Rush Medical College; Attending Physician, Cook County and St. Luke's Hospitals

Diseases of the Pleura

HENRY A. CHRISTIAN, A.M., M.D., LL.D.
Hersey Professor of the Theory and Practice of Physic, Harvard University, Physician-in-Chief to the Peter Bent Brigham Hospital, Boston

Acute Myocarditis and the Heart in Acute Infections, Healed Endocarditis (Valvular Disease of the Heart), Acute and Chronic Myocarditis

SAMUEL T. DARLING, M.D., D.Sc.
Formerly Resident Pathologist, Mercy Hospital, Baltimore; Chief of Laboratory, Ancon Hospital, Canal Zone; Investigation of Disease on the Rand Mines, South Africa (Gorgas Commission)

Tropical Diseases

FREDERICK FORCHHEIMER, M.D., Sc.D.
Formerly Professor of Medicine, Ohio-Miami Medical College, University of Cincinnati; Physician to Cincinnati Hospital

Mechanical and Circulatory Lesions of the Kidneys

SAMUEL J. GOLDBERG, M.D.
Attending Physician to the Grace Hospital, New Haven

Thrombo-Angiitis Obliterans

MALCOLM GOODRIDGE, M.D.
Professor of Clinical Medicine, Cornell University Medical College, Attending Physician, Bellevue Hospital

Arteriosclerosis

CHARLES LYMAN GREENE, M.D.
Formerly Attending Physician St. Luke's Hospital, Consulting Physician Minnesota State Hospital for Crippled Children; Professor of Medicine, Chief of Department of Medicine and Medical Clinic, University of Minnesota

Diseases of the Muscles

JAMES B. HERRICK, A.B., A.M., M.D.
Professor of Medicine, Rush Medical College, University of Chicago, Attending Physician Presbyterian Hospital, Chicago

Diseases of the Coronary Arteries, Angina-Pectoris—Cardiac Pain, Cardiac Neuroses

CHEVALIER JACKSON, M.D., Sc.D., F.A.C.S.
Professor of Bronchoscopy and Esophagoscopy, Jefferson Medical College, Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, University of Pennsylvania, Lecturer on Bronchoscopy and Esophagoscopy, Woman's Medical College

Foreign Bodies in the Bronchi

HENRY JACKSON, JR., A.B., M.D.
Assistant, Thorndike Memorial Laboratory, Boston City Hospital, Assistant Visiting Physician, Boston City Hospital

Diseases of the Bones

HOWARD SHEFFIELD JECK, Ph.B., M.D., F.A.C.S.
Instructor in Surgery, Department of Urology, Cornell University Medical College, Assistant Visiting Urologist, Bellevue Hospital

Cystitis, Tumors of the Bladder

CHARLES GODWIN JENNINGS, M.D.
Formerly Professor of Pediatrics and Practice of Medicine, Detroit College of Medicine and Surgery

Neuroses of the Bladder

EDWARD L. KEYES, M.D., Ph.D., F.A.C.S.
Professor of Urology, Cornell University Medical College, Urologist to St. Vincent's and Bellevue Hospitals

Cystitis

C. GUY LANE, A.B., M.D.
Assistant Dermatologist, Massachusetts General Hospital, Assistant in Dermatology, Courses for Graduates, Harvard Medical School

Occupational Dermatoses

A. J. LANZA, M.D.
Executive Officer, National Health Council; Formerly in Charge of Bureau of Industrial Hygiene of the United States Public Health Service and Chief Surgeon of the United States Bureau of Mines

Pneumoconiosis

EMANUEL LIBMAN, M.D.
Professor of Clinical Medicine, Columbia University, Physician to Mt. Sinai Hospital, New York City

Acute and Subacute Endocarditis

THOMAS McCRAE, M.D., F.R.C.P. (London)
Professor of Medicine, Jefferson Medical College, Physician to the Jefferson and Pennsylvania Hospitals

Syphilitic Aortitis and Aortic Aneurysm

ALEXANDER McPHEDRAN, M.D., LL.D.
Professor Emeritus of Medicine, University of Toronto, Consulting Physician to Toronto General Hospital and Hospital for Sick Children

Diseases of the Pericardium

WILLIAM FLETCHER McPHEDRAN, B.A., M.D.
Senior Demonstrator in Medicine, University of Toronto, Assistant Attending Physician, Toronto General Hospital

Diseases of the Pericardium

FRANK S. MEARA, A.B., Ph.D., M.D.
Professor of Clinical Medicine, Cornell University Medical College, Consulting Physician to Bellevue Hospital; Formerly Professor of Therapeutics, Cornell University Medical College

Essential Hypertension, Pyelitis, Hydronephrosis, Nephrolithiasis, Tuberculous Kidney

JOSEPH L. MILLER, B.S., M.D.
Professor of Medicine, Rush Medical College, Attending Medical Staff, Cook County and St. Luke's Hospitals

Nephritis, Amyloid Disease

HIDEYO NOGUCHI, M.D., M.S., Ph.D. (Imp. Univ. Tokyo), Sc.D.
Member Rockefeller Institute for Medical Research

Yellow Fever and Specific Prophylaxis of Yellow Fever

ALFRED TOWNSEND OSGOOD, M.D.
Professor of Genito-Urinary Surgery in New York University and Bellevue Hospital Medical College

Pyelitis, Hydronephrosis, Nephrolithiasis, Tuberculous Kidney

CLARENCE A. PATTEN, M.D.
Associate in Neurology, Graduate School of Medicine, University of Pennsylvania

Occupational Neuroses

PHILIP H. PIERSON, B.A., M.D.
Assistant Clinical Professor in Medicine, Stanford University Medical School, Chief of Chest Clinic, Lane Hospital

Diseases of the Lungs

J. F. ROGERS, M.D., Dr.P.H.
Chief, Division of Physical Education and School Hygiene, Bureau of Education, Department of Interior; Formerly Member of the Division of Industrial Hygiene and Sanitation of the United States Public Health Service

Occupational Intoxications

ROBERT DAWSON RUDOLF, C.B.E., M.D. (Edin.), F.R.C.P. (Lond.)
Professor of Therapeutics, University of Toronto, Clinician, Toronto General Hospital, Consulting Physician, Victoria Hospital for Sick Children and Toronto Convalescent Home

Diseases of the Bronchi

GEORGE ELMER SHAMBAUGH, Ph.B., M.D.
Professor of Otology and Laryngology, Rush Medical College, Otologist and Laryngologist to Presbyterian Hospital, Chicago

Diseases of the Nose and Accessory Sinuses

BURT RUSSELL SHURLY, B.S., M.D., F.A.C.S.
Chief of Staff and Teaching Clinic, Detroit Eye, Ear, Nose and Throat Hospital, Professor of Laryngology and Otology, Detroit College of Medicine

Diseases of the Nose and Accessory Sinuses, Diseases of the Larynx

RICHARD PEARSON STRONG, Ph.B., M.D., Sc.D.
Professor of Tropical Medicine, Harvard Medical School, Boston

Tropical Diseases

W. GILMAN THOMPSON, Ph.B., M.D.
Emeritus Professor of Medicine, Cornell University Medical College, President of the Reconstruction Hospital, Consulting Physician, Bellevue and Woman's Hospitals

Industrial Infections

THEODORE H. WEISENBURG, M.D.
Professor of Neurology, Graduate School of Medicine, University of Pennsylvania

Occupational Neuroses

CREIGHTON WELLMAN, M.D.
Formerly Professor and Head of the Department of Tropical Medicine, Hygiene and Preventive Medicine, and Director of the Laboratories of Hygiene and Tropical Medicine, Medical Department, Tulane University of Louisiana, Visiting Physician, Charity Hospital, New Orleans

Tropical Diseases

WILLIAM BUCHANAN WHERRY, A.B., M.D.
Associate Professor of Bacteriology, University of Cincinnati

Tropical Diseases

PAUL DUDLEY WHITE, A.B., M.D.
Instructor in Medicine, Harvard Medical School, Associate in Medicine and in Charge of Cardiac Clinics, Massachusetts General Hospital

Disorders of the Cardiac Mechanism

PAUL GERHARDT WOOLLEY, B.S., M.D.
Professor of Pathology, University of Cincinnati, Director of the Laboratories of the Cincinnati Hospital; Formerly Pathologist and Director of the Serum Laboratory, Bureau of Science, Manila, P. I.

Tropical Diseases

WADE WRIGHT, B.S., M.D.
In Charge of the Industrial Clinic, Massachusetts General Hospital, Instructor in Industrial Medicine, Harvard School of Public Health

Occupational Diseases Due to Physical Agencies

CONTENTS

DISEASES OF THE RESPIRATORY SYSTEM

CHAPTER I

DISEASES OF THE NOSE AND ACCESSORY SINUSES

Burt R. Shurly and George E. Shambaugh

PAGE
Relation of General Therapeutics to Special Treatment of the Upper Respiratory Tract . . . 3
Rhinitis . . . 4
  Acute Rhinitis . . . 4
  Chronic Rhinitis . . . 6
  Membranous Rhinitis . . . 7
  Atrophic Rhinitis . . . 8
Rhinoscleroma . . . 10
Sinusitis . . . 11
  Acute Nasal Sinusitis . . . 11
  Chronic Nasal Sinusitis . . . 13
  Hyperplastic Ethmoiditis . . . 16
Epistaxis . . . 17
Ozena . . . 19

CHAPTER II

DISEASES OF THE LARYNX

Burt R. Shurly

Laryngitis . . . 20
  Acute Laryngitis . . . 20
  Chronic Laryngitis . . . 22
  Edematous Laryngitis . . . 23
  Spasmodic Laryngitis . . . 23
  Membranous Laryngitis . . . 24
  Chronic Atrophic Laryngitis . . . 25
Tuberculosis of the Larynx . . . 25
Syphilis of the Upper Respiratory Tract . . . 30

CHAPTER III

DISEASES OF THE BRONCHI

Robert D. Rudolf

Bronchitis . . . 32
 Acute Bronchitis . . . 32
  General Therapy . . . 32
  Environment . . . 33

PAGE
Diet 36
Specific Treatment 36
Symptomatic Therapy 37
Local Treatment 38
Internal Medication 41
Measures for Relief of Cyanosis 45
Emetics 46
Chronic Bronchitis 47
Environment 48
Clothing 50
Contagion 50
Tobacco 50
Exercises 51
Diet and General Treatment 51
Vaccine Therapy 53
Serum Therapy 53
Symptomatic Treatment 54
Plastic Bronchitis 60
Treatment 60
Bronchiectasis 61
Environment 61
Diet 62
Specific Treatment 62
Surgical Treatment 63
Symptomatic Treatment 64
Bronchial Adenitis 67
Therapy 67
References 69

CHAPTER IV

FOREIGN BODIES IN THE BRONCHI

CHEVALIER JACKSON

Pathology of Foreign Bodies in the Bronchi 72
Arachidic Bronchitis 74
Lung-Suppuration 75
Symptomatology, Diagnosis, and Treatment of Foreign Body in the Air Passages 77
Symptomatology 77
Initial Symptoms 78
Laryngeal Foreign Body 78
Tracheal Foreign Body 79
Bronchial Foreign Body 80
Diagnosis 81
The Roentgen Ray 81
Physical Signs 81
Treatment 85
Coughing Up Foreign Bodies 85

PAGE
Disintegration of Foreign Bodies 86
Thoracotomy for Foreign Body 86
Treatment after Bechic Expulsion of a Bronchial Foreign Body . . 86
Bronchoscopic Removal of Foreign Bodies from the Lungs . . . 86
Bronchoscopic Removal of Penetrating Projectiles 88
References 88

CHAPTER V

DISEASES OF THE LUNGS

PHILIP H. PIERSON AND GEORGE BLUMER

Circulatory Disturbances 89
  Active Congestion 91
    Treatment 91
  Passive Congestion 92
    Treatment 94
  Hypostatic Congestion and Pneumonia 94
    Treatment 94
  Pulmonary Edema 95
    Etiology 95
    Theories as to Pathology 96
    Symptoms and Physical Signs 96
    Preventive Treatment 97
    Large Emboli 99
    Small Emboli 99
Bronchopneumonia 101
    Symptoms 102
    Prophylaxis 102
    Prophylactic Vaccination 103
    Specific Treatment 103
    Hygiene 105
    Fever 106
    Cyanosis and Dyspnea 107
    Distention 108
    Circulatory Collapse 108
    Complications 109
    Convalescence 110
Chronic Pneumonia 110
    Symptoms and Physical Signs 111
    Prophylactic Treatment 112
    Active Treatment 112
Pulmonary Emphysema 113
    Prophylaxis 114
    Freund's Theory and Operation 115
    Treatment 116
    Mechanical Devices 117
    Drugs 118

PAGE
Gangrene and Lung Abscess 119
  Gangrene 119
  Lung Abscess 120
    Location 121
    Symptoms and Physical Signs 122
    Medical Treatment 123
    Surgical Treatment 124
    Results of Treatment 125
    Postoperative Treatment 125
Tumors of the Lung 125
References 129

CHAPTER VI

DISEASES OF THE PLEURA

JOSEPH A. CAPPS

    Historical 134
    Thoracentesis 134
Pleurisy 136
    Clinical Pathology 136
    Absorption of Exudates 136
    Absorption of Fibrin 137
    Absorption of Fluid 137
    Absorption of Gases 138
    Permeability of the Mediastinum 138
    Mechanical Effect of Exudate 139
    The Intrathoracic Pressure of Exudates 139
    Influence of Exudates on Circulation 139
    Cause of Death from Pleural Exudate 140
    Pain Sense in the Pleura 140
    Onset of Pleurisy 142
    Clinical Course 142
    End Result after Absorption 143
    Treatment 143
    General Indications 143
  Fibrinous Pleurisy 144
    Acute Fibrinous Pleurisy 145
    Serofibrinous Pleurisy 145
    Treatment of the Infection 146
    Symptomatic Treatment 146
    To Check Secretion and Aid Absorption of Effusion 147
    Operative Measures 149
    Indications 149
    Other Considerations 150
    Choice of Time for Operations 150
    Contra-indications 151
    Location of Puncture 151

PAGE
Position of Patient 152
Preparation 152
Choice of Instrument 153
Method of Evacuation 153
Untoward Symptoms During Thoracenteses 156
Dangerous Symptoms and Their Treatment 157
Collapse and Sudden Death from Thoracentesis and Irrigation of Pleural Cavity 158
Intrapleural Injections to Aid Absorption 160
Autoserotherapy 161
Chronic Serous Effusion 162
After Treatment 162
Chylothorax 163
Hemothorax 163
Hemorrhagic Pleurisy 163
Traumatic Form 163
Pneumothorax 164
Accidental Pneumothorax during Thoracentesis 164
Pneumothorax Acutissimus 164
Traumatic Pneumothorax 165
Spontaneous Pneumothorax 165
Tuberculous Pneumothorax 165
Pneumothorax with Serous Effusion 165
Pneumothorax with Empyema 165
Artificial Pneumothorax for Therapeutic Purposes 166
Tumors of the Pleura 166
Treatment 167
Echinococcus Cysts 167
References 167

CHAPTER VII

DISEASES OF THE MEDIASTINUM

George Blumer

Mediastinal Tumors 172
Curative Treatment 172
Palliative Treatment 174
Mediastinitis 177
Suppurative Mediastinitis 177
Tuberculosis of the Mediastinal Lymph Nodes 178
Prophylaxis 178
Treatment 179
Indurative Mediastinopericarditis 181
Surgical Treatment 181
Technic 181
Results of Treatment 182
References 182

DISEASES OF THE CIRCULATORY SYSTEM

CHAPTER VIII

DISEASES OF THE PERICARDIUM

ALEXANDER McPHEDRAN AND WILLIAM FLETCHER McPHEDRAN

PAGE
- History . . . 187
- Pericarditis . . . 188
  - Treatment . . . 190
  - Diet in Acute Pericarditis . . . 191
  - Local Treatment . . . 193
  - Drugs . . . 194
  - Stimulants . . . 196
  - After Treatment . . . 197
  - Pericarditis with Effusion . . . 197
    - Paracentesis . . . 198
  - Hydropericardium . . . 201
  - Pyopericardium . . . 203
  - Hemopericardium . . . 203
  - Pneumopericardium . . . 204
  - Adhesive Pericarditis . . . 204
- References . . . 206

CHAPTER IX

ACUTE AND SUBACUTE ENDOCARDITIS

EMANUEL LIBMAN

- Subacute Bacterial Endocarditis . . . 207
  - Symptoms and Pathology . . . 207
    - Myocardial Symptoms . . . 208
    - Fever . . . 208
    - Anemia . . . 209
    - Emboli . . . 209
    - Petechiæ . . . 209
    - Pain and Tenderness . . . 209
    - Spleen . . . 210
    - Kidney . . . 210
    - Blood . . . 210
  - Diagnosis . . . 210
    - Bacterial Stage . . . 210
    - Bacteria-free Stage . . . 210
  - Treatment . . . 212
    - Vaccines and Serums . . . 212
    - Chemical Antidotes . . . 213
    - Transfusions . . . 214
    - Treatment of the Myocardial Symptoms . . . 215

PAGE
General Measure and Treatment of Toxemic Symptoms . . . 216
Treatment of the Embolic Phenomena 216
Treatment of the Bacteria-free Cases 217
Treatment of Focal Infections 217
Surgical Relations 217
Prophylaxis 218
Acute Bacterial Endocarditis 219
Etiology and Pathology 219
Symptoms 220
Course 221
Treatment 221

CHAPTER X

ACUTE MYOCARDITIS AND THE HEART IN ACUTE INFECTIOUS DISEASES

HENRY A. CHRISTIAN

Introduction 222
Symptomatology 223
Treatment 224
References 225

CHAPTER XI

TREATMENT OF CHRONIC MYOCARDITIS AND CHRONIC VALVULAR DISEASE OF THE HEART

HENRY A. CHRISTIAN

Introduction 227
Pathological Physiology 227
Etiology 232
Symptomatology 233
Treatment 233
Treatment During the Stage of Heart Failure or Decompensation . . 233
Rest 234
Digitalis 236
Other Drugs with Digitalislike Action 251
Other Drugs Acting on the Circulation 251
Drugs for Restlessness and Insomnia 251
Catharsis 252
Diuresis 254
Mechanical Removal of Fluid 255
Bleeding 256
Diet 256
Continuation Treatment 257
Getting the Patient Out of Bed 257
Continued Digitalis 258
Permissible Activity and Exercise 259
Diets 259
Management When Symptoms Are Absent or Slight 260

PAGE
Prevention of Cardiac Disease 261
Treatment of Special Conditions Occurring in Chronic Cardiac Disease 262
Auricular Fibrillation 262
Auricular Flutter 264
Extrasystoles 264
Heart-block 265
Paroxysmal Tachycardia 265
Pulsus Alternans 265
Chronic Myocarditis 265
High Blood-pressure 265
Aortic Insufficiency 266
Angina Pectoris 266
References 266

CHAPTER XII

ANGINA PECTORIS, CORONARY ARTERY DISEASE, CARDIAC PAIN

JAMES B. HERRICK

Angina Pectoris 272
Theories 272
Symptoms and Diagnosis 274
Prognosis 275
Prophylaxis 276
Treatment 276
Active Treatment 277
Treatment During an Attack 278
Treatment in the Intervals between Attacks 279
Drugs 282
Pseudo-Angina Pectoris 284
Treatment 284
Acute Coronary Obstruction 285
Treatment 287
Cardiac Pain 288
Pericarditis 289
Acute Myocarditis 290
Valvular Heart Disease 291
Paroxysmal Tachycardia 291
References 292

CHAPTER XIII

TREATMENT OF DISORDERS OF THE HEART BEAT

PAUL DUDLEY WHITE

Introduction 293
Sinus Arhythmia, Sinus Bradycardia, Sinus Tachycardia . . . 293
Sinus Arhythmia 293
Sinus Bradycardia 294
Sinus Tachycardia 294

PAGE
Premature Contractions 295
  Treatment 296
Paroxysmal Tachycardia 297
  General Treatment 297
  Drugs 298
Auricular Flutter 299
Auricular Fibrillation 301
  Digitalis Therapy of Auricular Fibrillation 302
  Quinidin Therapy of Auricular Fibrillation 305
Heart-Block 308
  Treatment 309
Pulsus Alternans 310
  Treatment 311

CHAPTER XIV

CARDIAC NEUROSES

JAMES B. HERRICK

The Irritable Heart 312
  Symptoms 314
  Diagnosis 315
  Prognosis 316
  Treatment 317
References 320

CHAPTER XV

THE TREATMENT OF CONGENITAL CARDIAC DISEASE

MAUDE E. ABBOTT

Introductory 322
Pathogenesis 325
  Congenital Cyanosis 325
  Primary Cause 326
  Secondary Causes 328
  The Cyanose Tardive of the So-called Non-cyanotic Group . . 330
Clinical Classification of Cardiac Defects 332
Treatment 336
 Prenatal Care 336
  Prenatal Infections and Intoxications 338
  Preventive Therapy 339
 Treatment of the Cyanotic Group 342
  Symptomatic Treatment of the Cyanotic Group 342
  Intravascular Oxygen Administration 346
  Preventive Treatment of the Cyanotic Group 346
 Treatment of the So-called Non-cyanotic Group 348
  Special Diagnostic Methods 355
  Estimation of Cardiac Tolerance 356
 Surgical Treatment 357
References 359

CHAPTER XVI

THE TREATMENT OF ARTERIOSCLEROSIS

MALCOLM GOODRIDGE

PAGE

- Definition . . . 365
- Types . . . 365
- Etiology . . . 365
- Pathology . . . 366
- Symptoms . . . 367

Treatment . . . 368
  - General Management . . . 368
    - Diet . . . 368
    - Habits . . . 369
    - Bowels . . . 369
    - Exercise . . . 369
    - Massage . . . 370
    - Hydrotherapy . . . 370
    - Electricity . . . 370
    - Radium . . . 371
    - Occupation . . . 371
    - Rest and Sleep . . . 371
  - Medicinal Treatment . . . 372
  - Symptomatic Treatment . . . 373
    - Coronary Sclerosis . . . 373
    - Renal Sclerosis . . . 374
    - Cerebral Arteriosclerosis . . . 374
    - Obliterating Endarteritis . . . 375
    - Intermittent Claudication . . . 376
  - Treatment of Special Types of Arteriosclerosis . . . 376
    - Senile Type . . . 376
    - Toxic or Infectious Type . . . 377
    - Hypertensive Type . . . 377

CHAPTER XVII

ESSENTIAL HYPERTENSION

FRANK SHERMAN MEARA

- Introduction . . . 378
- Results of Continued Hypertension . . . 379
- Treatment of Early Cases . . . 382
- Treatment of Later Cases . . . 382
- Hyperpietics with Low Diastolic Pressure . . . 384
- Patients with High Diastolic Pressure . . . 385
- Rest . . . 385
- Treatment of Cases without Symptoms . . . 385
- Treatment of Cases Showing Symptoms . . . 385
- Habits of Life . . . 386

PAGE
- Clothing 386
- Bathing 387
- Exercise 387
- Diet 387
- Summary 390

Cardiac Involvement 391
- Early Cases of Cardiac Insufficiency 391
- Dyspneas of Acute Onset 392
- Pulmonary Edema 394
- Pulmonary Infarcts 395
- Angina Pectoris 395
  - Treatment of Angina 398
  - Treatment of Acute Angina 399
  - General Treatment of Angina 401
  - Drugs 403
- Coronary Thrombosis and Embolism 403
  - Treatment 404
- High Blood-pressure Stasis 404
  - Treatment 404
- Auricular Fibrillation 405
  - Treatment 406
- Cardiac Failures with Edema 407
  - Treatment 408
  - Removal of Fluid 409
- Cardiac Murmurs 410
- Paroxysmal Tachycardia 410
  - Treatment 411
- Premature Systoles 412
  - Treatment 412

Vascular Accidents 412
- Cerebral Vessels 412
  - Treatment 413
- Retinal Vessels 415
- Thrombosis 415
  - Mesenteric Thrombosis 415
  - Thrombosis of Peripheral Vessels 416
- Hemorrhages 416
  - Treatment 416
- Erythrocytosis 417
- Anemia 417

Renal Lesions 418
- Uric Acid 420
- Urea Nitrogen 420
- Creatinin 421
- Salt 421
- Sugar 421
- Phenolsulphonephthalein 421

PAGE
Acidosis 422
  Treatment 422
Uremia 423
  Treatment of Uremia 425
  Treatment of the Convulsions 428
  Suppression of Urine 428
  Diet 428
Terminal Infection 429
Conclusion 430
  Summary of Treatment 430
  Cases Showing No Symptomatology 430
  Cases Showing A Symptomatology 430

CHAPTER XVIII

TREATMENT OF SYPHILITIC AORTITIS AND AORTIC ANEURYSM

THOMAS MCCRAE

Syphilitic Aortitis 439
  Clinical Features 439
  Treatment 439
  Acute Syphilitic Aortitis 440
  Chronic Aortitis 441
Aortic Aneurysm 441
  Pressure Signs 441
  Treatment 442
Thoracic Aneurysm 443
  Treatment 443
  Symptomatic 444
  Surgical Measures 444
  Heart 445
  Subsequent Management 445

CHAPTER XIX

THROMBOSIS, EMBOLISM AND PHLEBITIS

HARLOW BROOKS

Thrombosis 446
  Treatment 447
  Cardiac Thrombosis 449
  Pulmonary Thrombosis 449
  Mesenteric Thrombosis 450
  Peripheral Thrombosis 450
Embolism 450
  General Treatment 451
  Preventive Treatment 451
  Treatment of Fat Embolism 454
  Embolism of the Extremities 455
  Aortic Embolism 456

PAGE
- Cardiac Embolism 456
- Embolism of the Coronary Arteries 456
- Pulmonary Embolism 457
- Cerebral Embolism 457
- Splenic and Renal Embolism 458
- Embolism of the Mesenteric Vessels 458

Phlebitis 458
- Non-obliterative Phlebitis 461
- Obliterative Phlebitis 461
- Recurrent Phlebitis 462
- Chronic Phlebitis 462
- General Treatment 463
- Surgical Treatment 467

References 468

CHAPTER XX

THROMBO-ANGIITIS OBLITERANS

S. J. GOLDBERG

- Synonyms 469
- Definition 469
- Etiology 469
- History and Geographical Distribution 470
- Incidence (Age, Sex and Race) 471
- Pathology 472
- Symptoms and Diagnosis 472
- Prognosis 474
- Treatment 475
- Drugs 475
- Heat and Elevation of Member 475
- Electrotherapy 476
- Bier Suction Cylinders 476
- Femoral Vein Ligation and Arteriovenous Anastomosis . . . 476
- Intravenous and Subcutaneous Injections 477
- Leriche's Periarterial Sympathectomy 478

CHAPTER XXI

DISEASES OF THE MUSCLES

CHARLES LYMAN GREENE

Various Forms of Myositis 483
- Acute Polymyositis 483
- Syphilis of the Muscles 484
- Primary Acute Suppurative Myositis 485
- Secondary Suppurative Myositis 486
- Tuberculous Myositis 486
- Myositis Fibrosa 486

PAGE

  - Myositis Ossificans 486
  - Treatment 487
- Muscular Rheumatism (Myalgia) 487
  - Treatment 488
- Muscular Cramps 489
- Localized Rupture of the Muscles 489
  - Treatment 490
- References 490

CHAPTER XXII

DISEASES OF THE BONES

HENRY JACKSON, JR.

  - Synonyms 492
  - Definition 492
  - Etiology 492
  - Pathology 492
  - Symptoms 492
  - Treatment 493
- Secondary Hypertrophic Osteoarthropathy 493
  - Definition 493
  - Etiology 493
  - Pathology 494
  - Symptoms 494
  - Treatment 494
- Osteomalacia 494
  - Definition 494
  - Etiology 494
  - Pathology 495
  - Clinical Forms 495
  - Symptoms 495
  - Course and Progress 495
  - Treatment 495
- References 496

CHAPTER XXIII

MECHANICAL AND CIRCULATORY LESIONS OF THE KIDNEYS

FREDERICK FORCHHEIMER

- Movable Kidney 501
- Anuresis 503
  - Treatment 503
- Hematuria 504
- Hemoglobinuria 504
  - Toxic Hemoglobinuria 504
  - Treatment 506
- Paroxysmal Hemoglobinuria 506
  - Treatment 507

CHAPTER XXIV

NEPHRITIS

Joseph L. Miller

PAGE
Introduction 508
Normal Physiology of the Kidney 508
Theories of Renal Secretion 508
Composition of Urine 510
Origin and Significance of Urea 510
Internal Secretion of the Kidneys 511
Pathological Physiology of the Kidneys 512
Edema 513
Summary 515
Increased Blood-pressure 516
Cardiac Hypertrophy 517
Uremia 518
Summary 519
Value of Functional Kidney Tests 520
Principles of Treatment of Nephritis 522
Prophylaxis 523
Climate 526
Rest 527
Bathing and Care of the Skin 528
Elimination through the Bowel 528
Restriction of Fluids in Nephritis 530
Restriction of Sodium Chlorid 531
Summary 532
Sweating 533
Summary 535
Diet 636
Summary 540
Treatment of Hypertension 540
Summary 544
Use of Diuretics 544
Summary 547
Treatment of Uremia 547
Uremic Dyspnea and Pulmonary Edema 550
Summary 551
Special Treatment 551
Acute Nephritis 551
Prophylaxis 552
Active Treatment 553
Summary 559
Chronic Parenchymatous Nephritis 559
Elimination 560
Diuretics 560

PAGE
Diet 560
Summary 561
Chronic Interstitial Nephritis 561
Diet 562
Salt Restriction 562
Hypertension 563
Dyspnea 564
Headache 565
Cerebral Hemorrhage 565
Summary 566
Mercurial Nephritis 566
References 567

CHAPTER XXV

AMYLOID DISEASE OF THE KIDNEY

JOSEPH L. MILLER

The Kidney of Pregnancy 577
Reference 578

CHAPTER XXVI

PYELITIS

FRANK SHERMAN MEARA AND ALFRED TOWNSEND OSGOOD

Treatment 581
Diet 582
Local Treatment 583
Bowels 584
Diuretics and Urinary Antiseptics 584
Vaccine Therapy 586
Operation 587
Complications 589
Cystitis 589
Retention of Urine 589
Suppression of Urine 589
Peritonitis 589
Cardiac Hypertrophy and Hypertension 589
Gastro-intestinal Symptoms 589
Prophylaxis 590
References 591

CHAPTER XXVII

HYDRONEPHROSIS

FRANK SHERMAN MEARA AND ALFRED TOWNSEND OSGOOD

Treatment 593
References 595

CHAPTER XXVIII

NEPHROLITHIASIS

FRANK SHERMAN MEARA AND ALFRED TOWNSEND OSGOOD

PAGE
Treatment 597
  Renal Colic 597
    Cold 599
    Enteroclysis 599
    Warm Drinks 599
    Posture 600
    Drugs 600
  Treatment of Other Symptoms Than Pain During the Attack . . 602
  Lithiasis 604
    Exercise 604
    Diet 604
  Renal Calculus 606
References 607

CHAPTER XXIX

TUBERCULOUS KIDNEY

FRANK SHERMAN MEARA AND ALFRED TOWNSEND OSGOOD

Treatment 608
    Diet 612
    Tuberculin 613
    Symptomatic Treatment 613
    Complications 614
    Prophylaxis 614
References 614

DISEASES OF THE BLADDER

CHAPTER XXX

CYSTITIS

EDWARD L. KEYES AND HOWARD S. JECK

Prophylaxis 617
    Prevention of Gonorrheal Cystitis 617
    Prevention of Infection by the Tubercle Bacillus 617
    Prevention of Spontaneous Pyogenic Infections 617
    Prevention of Catheteral Infection 618
Treatment 618
    Acute Cystitis 618
    Chronic Cystitis 618
    Tuberculous Cystitis 619
    Hunner Ulcer 619

PAGE
Functional Neuroses of the Bladder 619
  Treatment of Enuresis 619
  Treatment of Reflex Neuroses of the Bladder 620
  Paralysis of the Bladder 621

CHAPTER XXXI

NEUROSES OF THE BLADDER

CHARLES GODWIN JENNINGS

  Paralysis of the Bladder 622
Enuresis 623
  Treatment 624
  Hygienic 625
  Urinary Changes 625
  Diseases of the Urinary and Pelvic Organs 626
  Diseases of Remote Organs 628
  General Treatment 630
  Drugs 631

CHAPTER XXXII

THE TREATMENT OF TUMORS OF THE BLADDER

HOWARD S. JECK

  Tumors of Epithelial Origin 633
  Tumors of Connective Tissue Origin 633
Symptoms 633
  Dysuria 634
  Pain 634
  Retention 634
  Cystitis 634
  Constitutional and Renal Symptoms 634
Non-operative Control of Hemorrhage from Bladder Tumors . . . 635
Methods of Treatment 636
Fulguration 636
  Technic of Fulguration 637
Operation 638
  Suprapubic Cystotomy 638
  Excision of Bladder Tumors and Destruction by Cautery . . . 643
Radiation 643
  Radium Emanation 643
  Technic in Cases Suitable for Cystoscopic or Urethroscopic Treatment 643
  Implantation of Radium Seeds after Exposure of the Growth by Suprapubic Cystotomy 648
  Deep X-ray Therapy 648
  Cystectomy, Ureteral Transplantation, Cystostomy 648
Treatment Best Suited to a Particular Type of Growth 650
References 650

OCCUPATIONAL DISEASES

CHAPTER XXXIII

INDUSTRIAL INFECTIONS

W. GILMAN THOMPSON

PAGE
Comparison of Industrial and Other Infections 653
Extrahazardous Industries 654
Prevention 656

CHAPTER XXXIV

INDUSTRIAL INTOXICATIONS

JAMES FREDERICK ROGERS

- Introduction 661
- Entry and Exit 661
- Pathology and Symptoms 661
- Diagnosis 662
- Prevention 662
- Treatment 663

Metals and Their Compounds 663
- Antimony 663
  - Pathology 663
  - Symptoms 663
  - Prevention 664
  - Treatment 664
- Arsenic 664
  - Pathology 664
  - Symptoms of Acute Poisoning 664
  - Chronic Poisoning 665
  - Prevention 665
  - Treatment 665
- Brass, Zinc, Etc. 665
  - Symptoms of Acute Poisoning 666
  - Diagnosis 666
  - Prevention 667
  - Treatment 667
  - Chronic Poisoning of Workers in Brass 667
  - Prevention 667
  - Treatment 667
- Lead 667
  - Pathology 668
  - Symptoms 668
  - Diagnosis 670
  - Prevention 670
  - Treatment 671

PAGE
Manganese 672
  Pathology 672
  Symptoms 672
  Prevention 672
  Treatment 672
Mercury 672
  Pathology 673
  Symptoms 673
  Prevention 674
  Treatment 674
Non-metals and Their Compounds 674
  Anilin and Its Homologues and Other Amino Compounds . . . 674
    Pathology 674
    Symptoms 674
    Diagnosis 675
    Prevention 675
    Treatment 675
  Benzene (Benzol), Benzin, Naphtha and Gasoline 675
    Pathology 676
    Symptoms 676
    Prevention 676
    Treatment 677
  Carbon Bisulphid 677
    Pathology 677
    Symptoms of Acute Poisoning 677
    Chronic Poisoning 677
    Prevention 678
    Treatment 678
  Carbon Monoxid 679
    Pathology 679
    Acute Poisoning 679
    Chronic Poisoning 679
    Prevention 680
    Treatment 680
  Carbon Tetrachlorid or Tetrachlormethane 680
Methyl Alcohol 681
    Pathology 681
    Symptoms 681
    Chronic Poisoning 681
    Prevention 681
    Treatment 681
  Nitrobenzol, Dinitrobenzol and Trinitrobenzol 681
    Pathology 681
    Symptoms 681
    Prevention 682
    Treatment 682

PAGE
Nitroglycerin 682
  Pathology 682
  Symptoms 682
  Prevention 683
  Treatment 683
Phosphorus and Phosphorated Hydrogen 683
  Pathology 683
  Symptoms 683
  Prevention 683
  Treatment 683
Sulphurated Hydrogen 683
  Pathology 683
  Symptoms 683
  Prevention 684
  Treatment 684
Tetrachlorethane or Tetralin 684
  Pathology 684
  Symptoms 684
  Prevention 685
Trinitrotoluene (TNT) 685
  Pathology 685
  Symptoms 685
  Prevention 686
  Treatment 686
Turpentine 686
  Pathology 686
  Symptoms 687
  Prevention 687
  Treatment 687
Miscellaneous 687
References 689

CHAPTER XXXV

OCCUPATIONAL DISEASES DUE TO PHYSICAL AGENCIES

Wade Wright and John W. S. Brady

Compressed Air Illness, or Caisson-Disease 690
  Predisposing Factors in Caisson-Disease 696
    Ventilation 699
    Selection of Workers 700
    Decompression 701
  Treatment of Caisson-Disease 704
Heat, Humidity and Cold 706
  Heat 706
    Heat Exhaustion and Heat Stroke 707
    Heat Cramps 709
    Miscellaneous Effects of Heat 709
    Prophylaxis 710

PAGE
Heat and Humidity . . . 710
Cold . . . 714
Prophylaxis and Treatment . . . 715
Light . . . 716
Glass Worker's Cataract . . . 718
Prophylaxis and Treatment . . . 719
Electric Ophthalmia . . . 720
Treatment . . . 721
Ultraviolet Rays . . . 721
Prophylaxis of Exposure to Ultraviolet Rays . . . 724
Treatment . . . 724
Eclipse Blindness . . . 725
Ocular Fatigue . . . 725
Prophylaxis . . . 726
Miner's Nystagmus . . . 727
Treatment . . . 729
Eye Strain and Poor Lighting . . . 729
Prophylaxis . . . 732
Radiation . . . 732
Radium . . . 736
Dermatitis . . . 738
Electricity . . . 741
Electrical Burns . . . 742
Treatment . . . 743
Electrical Shock . . . 743
Treatment . . . 744
Prophylaxis . . . 747
Electric Ophthalmia . . . 747
Lightning Shock . . . 747
Fatigue . . . 748
The Physiology of Fatigue . . . 748
Industrial Fatigue . . . 752
Output . . . 755
Hours of Labor . . . 761
Rest Periods . . . 763
Night Work . . . 764
Temperature, Humidity and Ventilation . . . 764
Lighting . . . 764
Occupational Neuroses . . . 766
References . . . 767

CHAPTER XXXVI

PNEUMOCONIOSIS

A. J. Lanza

Symptomatology and Prognosis . . . 773
Treatment and Prevention . . . 774
References . . . 775

CHAPTER XXXVII

OCCUPATIONAL NEUROSES

CLARENCE A. PATTEN AND THEODORE H. WEISENBURG

PAGE
Symptomatology 777
Treatment 780

CHAPTER XXXVIII

OCCUPATIONAL DERMATOSES

C. GUY LANE

General Considerations 782
Prophylaxis 783
Treatment 784
Acne 784
Etiological Factors 784
Prophylaxis 784
Treatment 785
Actinomycosis 785
Etiological Factors 785
Prophylaxis 785
Treatment 786
Anthrax 786
Etiological Factors 786
Prophylaxis 786
Treatment 786
Burns 786
Etiological Factors 786
Prophylaxis 787
Treatment 787
Callositas (Callus) 787
Etiological Factor 787
Prophylaxis 787
Treatment 788
Dermatitis (Acute Type) 788
Etiological Factors 788
Prophylaxis 788
Treatment 788
Eczema 789
Etiological Factors 790
Prophylaxis 790
Treatment 790
Epithelioma (Skin Cancer) 791
Etiological Factors 791
Prophylaxis 791
Treatment 791

PAGE
Folliculitis 791
    Etiological Factors 791
    Prophylaxis 791
    Treatment 792
Keloids 792
    Etiological Factors 792
    Prophylaxis 792
    Treatment 792
Malingering (Dermatitis Factitia and Artificial Eruption) . . . 793
    Prophylaxis 793
    Treatment 793
Paronychia 793
    Etiological Factors 793
    Prophylaxis 793
    Treatment 793
Pus Infections 794
    Etiological Factors 794
    Prophylaxis 794
    Treatment 794
Tinea (Ringworm) 795
    Etiological Factors 795
    Prophylaxis 795
    Treatment 795
Tuberculosis of the Skin 795
    Etiological Factors 795
    Prophylaxis 795
    Treatment 796
Ulcers 796
    Etiological Factors 796
    Prophylaxis 796
    Treatment 796

TROPICAL DISEASES

CHAPTER XXXIX

INTRODUCTION—THE RÔLE OF THE MEDICAL MAN IN THE FUTURE CONTROL OF THE TROPICS

William B. Wherry

References 803

CHAPTER XL

INSOLATION

Paul G. Woolley

    Prophylaxis 808
    Treatment 809
References 811

CHAPTER XLI

TROPICAL INFECTIONS OF BACTERIAL OR UNKNOWN ETIOLOGY

Samuel T. Darling, Richard P. Strong and Paul G. Woolley

PAGE
- Undulant Fever (Malta Fever) . . . 813
  - Treatment . . . 813
    - Serum and Vaccine Treatment . . . 813
    - Symptomatic Treatment . . . 814
- Dengue and Dengue-like Fevers . . . 814
  - Prophylaxis . . . 816
  - Treatment . . . 816
- Pappataci Fever . . . 817
  - Treatment . . . 817
  - Prophylaxis . . . 81
- Tsutsugamushi Fever . . . 81
  - Prophylaxis . . . 818
  - Treatment . . . 819
- Sprue . . . 819
  - Etiology . . . 820
  - Pathology . . . 820
  - Symptoms . . . 821
  - Prognosis . . . 821
  - Treatment . . . 821
  - Hill Diarrhea . . . 823
    - Treatment . . . 823
  - Low Country Morning Diarrhea . . . 823
- Verruga Peruviana . . . 824
  - Prophylaxis, and Nature of the Disease and Distribution . . . 824
  - Age, Sex, and Race . . . 825
  - Etiology . . . 826
  - Prophylaxis . . . 826
  - Treatment . . . 827
- Oroya Fever . . . 828
  - Prophylaxis . . . 828
  - Personal Prophylaxis and Treatment . . . 829
- Tropical Local Inflammations . . . 830
  - Ulcerating Granuloma of the Pudenda . . . 830
  - Tropical Ulcer . . . 832
  - Climatic Buboes . . . 833
- References . . . 833

CHAPTER XLII

TROPICAL DISEASES DUE TO PROTOZOA

Samuel T. Darling, Hideyo Noguchi, Creighton Wellman, William B. Wherry, and Paul G. Woolley

PAGE
The Spirochetoses 836
The Relapsing Fevers 836
  Prophylaxis 837
  Treatment and Medication 838
  Symptomatic Treatment 838
Frambesia Tropica 839
  Symptoms 841
  Treatment 841
  Prophylaxis 842
Yellow Fever 842
  General and Symptomatic Treatment 844
  Specific Treatment 844
Specific Prophylaxis of Yellow Fever 845
  Isolation of the Patient and Destruction of Infected Mosquitoes . . 845
  Personal Precautions 846
  Antistegomyia Measures 846
  Prophylactic Inoculation Against Yellow Fever 846
The Trypanosomiases 848
  Cause 848
  Pathology 849
  Symptomatology 849
  General Treatment 849
  Specific Treatment by Vaccine or Serum 850
  General Treatment 852
  Preventive Measures 852
  Drug Prophylaxis 855
Human Trypanosomiasis of South America (Chagas' Disease) . . . 856
  Treatment 857
  Prophylaxis 857
The Leishmaniases 858
 Visceral Leishmaniases 858
  Prophylaxis 859
  Treatment 860
 Dermal Leishmaniases 862
  Synonyms 862
  Prophylaxis 864
  Treatment 864
Diseases Due to Flagellata and Ciliata 866
  Balantidium Coli 866
References 867

CHAPTER XLIII

DISEASES DUE TO TROPICAL CESTODES, TREMATODES AND NEMATODES

Samuel T. Darling, Creighton Wellman and William B. Wherry

PAGE
Teniasis 870
Somatic Teniasis 870
Intestinal Teniasis 871
Distomiasis 874
Hepatic Distomiasis 874
Intestinal Distomiasis 875
Endemic Hemoptysis 877
Preventive Measures 877
Schistosomiasis 877
Intestinal Schistosomiasis 879
Katayama Disease 880
Filariasis 880
Filaria Bancrofti 880
Dracunculus Medinensis (Guinea Worm, Medina Worm, the Worm of Pharaoh) 882
Filaria Loa 883
Filaria Perstans 884
Other Nematode Infections 884
Strongyloides Stercoralis 884
Trichocephaliasis 886
Strongylosis 886
Esophagostomiasis 887
Other Nematodes of Doubtful Pathogenesis 888
Gnathostoma Siamense 888
Leeches 888
References 889

CHAPTER XLIV

AFFECTIONS DUE TO POISONOUS REPTILES, FISH, ARTHROPODS (INCLUDING MYIASIS) AND PLANTS

Creighton Wellman

Revised by Samuel T. Darling

Snake Poisoning 890
General and Specific Treatment 891
Non-Specific Treatment 891
Prophylaxis 892
Other Poisonous Vertebrates 892
Batrachia and Lacertilia 892
Poisonous Fish 893
Poisonous Mammals 894
Venomous Arthropods 894
Spider Bite 894

PAGE
Scorpion Bites 894
Centipede Bites 895
Poisoning by Ticks, Lice and Bugs 895
Tick and Louse Poisoning 895
Poisonous Insects—Flies (Myiasis), Bees, Wasps, and Ants (Lepidoptera) 896
Poisoning by Caterpillars, Butterflies and Moths 897
Treatment 897
Poisonous Flies and Fleas 897
Treatment 897
Myiasis 897
Treatment 898
The Jigger 900
Treatment 900
Prophylaxis 900
Atriplicism 900
Remedial Measures 901
Lathyrism 901
Remedial Measures 901
Arrow Poisoning 901
Strychnos 901
Strophanthus 902
Erythrophlœum 902
Tropical Intoxications 904
Poisons Used for Murder, Suicide, the Production of Abortion . . 904
Irritative Poisonous Plants 905
Prophylaxis 906
Treatment 906
Fish Poisoning 906
Treatment 906

LIST OF ILLUSTRATIONS

FOREIGN BODIES IN THE BRONCHI

Chevalier Jackson

FIGURE PAGE

1. Roentgenogram showing shawl pin in lung of a woman aged 41 years . . . 73
2. Roentgenogram showing a safety pin in the lung of a woman aged 46 years . . . 74
3. Roentgenogram showing brass paper fastener in the lung of a woman aged 23 years . . . 75
4. Seven years after peroral bronchoscopic removal of brass paper fastener that had been in the lung seven years . . . 76
5. Roentgenograms, anteroposterior and lateral, of a child aged 23 months showing pulmonary abscess caused by the unknown presence of a screw for 20 months . . . 77
6. A case of total absence of the cough reflex despite the presence during a period of 2 months, of a safety pin in the lung of a woman aged 18 years . . . 79
7. A case of absence of cough despite the present of a shawl pin deep in the left lower lobe (patient a girl aged 10 years) . . . 80
8. Roentgenogram showing in the right hand illustration a small electric lamp in left lung of a boy aged 6 years . . . 82
9. Expiratory valvelike bronchial obstruction by non-radiopaque foreign body, producing an acute obstructive emphysema . . . 82

DISEASES OF THE PERICARDIUM

Alexander McPhedran and William Fletcher McPhedran

1. The Gatch bed . . . 190

TREATMENT OF CHRONIC MYOCARDITIS AND CHRONIC VALVULAR DISEASE OF THE HEART

Henry A. Christian

1. Male, aged 28, chronic cardiac valvular disease, mitral stenosis; rhythm regular . . . 237
2. Female, age 28, chronic cardiac valvular disease, mitral stenosis and regurgitation, aortic regurgitation, auricular fibrillation . . . 238
3. Male, age 57, chronic myocarditis, auricular fibrillation . . . 239
4. Male, age 35, chronic myocarditis, regular rhythm and marked degree of Pulsus alternans . . . 240
5. Male, age 59, chronic myocarditis, auricular fibrillation, arteriosclerosis, old right hemiplegia . . . 241
6. Male, age 60, chronic myocarditis, rhythm regular . . . 242

FIGURE PAGE
7. Female, age 45, chronic myocarditis, auricular fibrillation . . . 243
8. Male, age 45, chronic myocarditis, hypertension, rhythm regular . 245
9. Female, age 43, chronic myocarditis, hypertension, rhythm regular . 247
10. Male, age 56, chronic myocarditis, rhythm regular 248
11. Patient with aortic stenosis and insufficiency 253
12. Same patient as in Figure 11 254

THE TREATMENT OF CONGENITAL CARDIAC DISEASE

Maude E. Abbott

1. Diagram showing the proportion of oxygenated hemoglobin to reduced hemoglobin in different parts of the circulatory system in normal resting individuals 327
2. Diagram showing the proportion of oxygenated to reduced hemoglobin in different parts of the circulatory system in a case of incomplete oxygenation in aërated parts of the lungs 327
3. Diagram showing the proportion of oxyhemoglobin to reduced hemoglobin in different parts of the circulatory system in a case where a fraction of blood passes through unaërated channels from venous to arterial system 328
4. Diagram showing the relative magnitude of the influence of mean capillary oxygen unsaturation, of variations in total oxygen combining power of the blood, in deoxidation rate during the passage of blood through the capillaries, in the fraction of total hemoglobin passing unoxidized through aërated parts of the lungs, and in fractions of blood passing from right to left through channels to which air has no access 329
5. Diagram showing influence of the mean capillary oxygen unsaturation of simultaneous variations in T & λ 330
6. Diagram showing the influence of the mean capillary oxygen unsaturation of simultaneous variations in D and α 331
7. Diagram showing the proportion of oxyhemoglobin to reduced hemoglobin in different parts of the circulatory system in a case where a fraction of blood passes from the left side of the heart to the right side through an abnormal communication 332
8. Diagrams showing inverse ratio in basal carrying tension and volume of blood in pulmonary circulation in septal defects . . . 333
9. Localized defect of the interventricular septum in two cases belonging through their anatomical configuration respectively to the (A) cyanotic, and (B) non-cyanotic group 335
10. Clubbing of toes in congenital cyanosis 343
11. Methods for administering oxygen to young infants 345
12A. To illustrate left to right shunt with terminal reversal of flow. Large defect at the upper part of the interauricular septum (persistent ostium secundum). Hypoplasia of the aorta and great dilatation of the pulmonary artery with marked thickening and calcification of the pulmonary valves. Hypertrophy and dilatation of right chambers 350

FIGURE PAGE

12B. To illustrate left to right shunt with terminal reversal of flow. Large defect at the upper part of the interauricular septum (persistent ostium secundum), hyperplasia of the aorta and great dilatation of the pulmonary artery with marked thickening and calcification of the pulmonary valves, hypertrophy, and dilatation of the right chambers 351

13. Patent ductus arteriosus with acute infective endarteritis about pulmonary orifice of ductus and multiple embolic abscesses of lung. Pneumococcus infection 352

14.A Aneurysm of the right aortic sinus of Valsalva rupturing into the conus of the right ventricle with defect of the anterior interventricular septum and malignant endocarditis of the margins of the defects in the right mural endocardium of the right ventricle adjacent and opposite these and of the pulmonary and aortic valves 353

14B. Aneurysm of the right aortic sinus of Valsalva rupturing into the conus of the right ventricle with defect of the anterior interventricular septum and malignant endocarditis of the margins of the defects in the right mural endocardium of the right ventricle adjacent and opposite these and of the pulmonary and aortic valves . . . 354

NEPHRITIS

Joseph L. Miller

1. Fluctuation in blood-pressure in a patient with hypertension . . 541

2. Comparative effect on blood-pressure of nitroglycerin (solid line) erythrol tetranitrates (line composed of dashes) and sodium nitrite (dotted line) 544

THE TREATMENT OF TUMORS OF THE BLADDER

Howard S. Jeck

1. Exposure of the bladder 639

2. Incision of the bladder wall 641

3. Implantation of radium "seeds" by means of the cystoscope . . 644

4. Application of unscreened radium emanation to surface of tumor by means of the cystoscope 645

5. Application of screened radium emanation to growth near bladder neck 646

6. Exposure of tumor for implantation of radium "seeds" under direct vision 647

7. Implantation of radium "seeds" after exposure of the tumor (infiltrating carcinoma) by suprapubic cystotomy 649

OCCUPATIONAL DISEASES DUE TO PHYSICAL AGENCIES

Wade Wright and John W. S. Brady

1. Percentage transmission of various glasses for infra-red radiation . 720

2. Variation of intensity of solar heat (dotted line) and intensity of ultraviolet radiation (solid line) throughout the day in different months—in terms of arbitrary units 722

FIGURE PAGE
3. Heat (H), brightness (B), and ultraviolet (U. V.) intensities at noon throughout the year at Davos, Switzerland 723
4. Reading the foot candle meter 731
5. Foot candle meter 731
6. Hourly output of workers at various types of processes . . . 757
7. Seasonal fluctuation of sickness rate 759
8. Seasonal variation in sickness rate for all diseases (light line) and all diseases exclusive of influenza and grippe (heavy line) . . 759
9. Frequency of sickness and non-industrial accidents over two-year period classified according to length of service with the employing company 760
10. Results of reduction of hours 763
11. Seasonal relations of temperature and output 765

TROPICAL DISEASES DUE TO PROTOZOA

Samuel T. Darling

1. Glossina palpalis 855

DISEASES DUE TO TROPICAL CESTODES

Samuel T. Darling

1. Schistosomal papilloma of the rectum 878

DISEASES OF
THE RESPIRATORY SYSTEM

CHAPTER I

DISEASES OF THE NOSE AND ACCESSORY SINUSES

Burt R. Shurly and George E. Shambaugh

RELATION OF GENERAL THERAPEUTICS TO SPECIAL TREATMENT OF THE UPPER RESPIRATORY TRACT

The natural modern trend of our highly developed specialties is rapidly producing a sharp line of demarcation between the treatment of general and special pathologic phenomena. There is danger in this development that rhinology and laryngology may become departments of therapy only, while the state of the general human machine is frequently ignored. Many disastrous failures of treatment by laryngologists are examples of this fact. Many conditions of the upper respiratory tract are wholly systemic in origin and must be treated on a broad and liberal basis. For example, this department of medicine must be ever alert to offer therapeutic care to the various special and frequently general manifestations that occur in syphilis, tuberculosis, diphtheria, diseases of the ductless glands, gout, rheumatism, and various general infections of the staphylococcus, streptococcus, influenza bacilli, and other pathogenic bacteria. Therapeutics has developed a series of specifics and other remedies of great value. Their usefulness is placed upon a definite and exact basis of value. These therapeutic indications must be met as promptly by the specialist as by the internist. Textbooks are laden with innumerable drugs and general therapeutic measures that are recognized by practitioners as worthless and harmful from the fact that frequently valuable time is lost in the treatment of disease by therapeutic methods that deserve a place only in the history of medicine. It has been our endeavor to eliminate and discard remedies that serve to fill the pages of the textbook and mention those only which clinical experience has proved of real curative merit, or at least as additions to the comfort, and not the discomfort, of the patient. There is a place in laryngology for the physician as well as the surgeon. Recently the radical surgeon laryngologist is receding to the more logical fields of conservatism, and slowly he will appreciate more and more the true usefulness of his friend and consultant, the physician laryngologist.

RHINITIS

Acute Rhinitis (*Acute Coryza*).—The treatment of acute rhinitis involves primarily a study of the habits, dress, occupation, temperament, local and general physical defects and immunity of the individual. This study will develop principles of prophylaxis that require enforcement by careful attention to details.

Neglect or carelessness in regard to clothing, house ventilation or heating, and exposure must be studied and the remedy suggested. A definite plan of cold sponge bathing or sleeping outdoors may be of prophylactic benefit. The neck and chest may be quickly sponged with cool and later very cold water, or the body may be gone over with a stiff flesh brush. When this latter procedure is adopted the smallest amount of water which will form a lather with pure white castile or common laundry soap should be used. A sharp reaction of the skin should be obtained afterward by brisk friction with a Turkish towel, which will remove the lather without rinsing. This morning bath should be taken in a room which is thoroughly warmed. In debilitated subjects the change to a cold bath should be made gradually.

Many individuals are particularly susceptible to drafts of air, and some to drafts when applied to definite areas of the body, such as the back of the neck, the interscapular region, or about the feet. Advice in regard to wearing apparel may be advantageously given. The use of high shoes, warm stockings, and proper underwear of wool, silk, or flannel will prove a necessary prophylaxis in some cases. Chest protectors, neck furs, and mufflers should be dispensed with. Many colds may be prevented by the prompt removal of damp clothing, especially shoes, so that body heat is quickly restored.

In cases of recurrent coryza the condition of the nasal and nasopharyngeal chambers must be thoroughly investigated. Septal deformities, hypertrophies, adenoids, polypi, chronic sinus infection and all other important conditions that interfere with nasal drainage and respiration must receive proper surgical attention. When acute coryza is present in the household delicate subjects may receive a prophylactic inoculation of a vaccine. This may be prepared from a nasal culture obtained from the member of the family who was the original source of infection.

A careful regulation of the temperature of living rooms to about 65° F. is important, while a cold, well-ventilated sleeping room, warm bed covering, and night garments are advantageous to many people during the winter months. Susceptible infants and young children may be provided with an improvised sleeping bag or blankets may be pinned to avoid the dangers of exposure at night. Sleeping out of doors or in a window tent has given excellent prophylactic results in selected cases.

Against an acute attack radical abortive measures must be adopted early. The time-honored administration at bedtime of 5 to 10 gr. (0.3 to 0.6 gm.) of Dover's powder alone, or with 5 gr. (0.3 gm.) each of aspirin and quinin, is frequently successful. A mustard foot bath will encourage leukocytosis; and a hot lemonade with spiritus frumenti will establish diaphoresis. The patient should immediately retire and remain covered with several warmed blankets until profuse perspiration ensues. On rising in the morning a saline laxative such as a Seidlitz powder or Sol. Rochelle ℥ss may be necessary. During the day the Lincoln formula of quinin sulphate, gr. ½ (0.03 gm.); fluid ext. of belladonna, gr. ⅛ (0.007 gm.); gum camphor, gr. ¼ (0.012 gm.), every two or three hours; or 3 drops each of tr. belladonna and tr. opium in a half ounce (15 c.c.) of camphor water may be given.

The nose should be examined under good illumination and a culture taken from the secretion if the vaccine treatment is to be adopted, or a stock vaccine may be chosen. If Bosworth's method be selected—then a 20 per cent solution of cocain is applied to the inferior turbinates, after which a bead of chromic acid is fused upon a probe, and with this a fine linear cauterization is made along the median surface of each inferior turbinate.

Trouble may be avoided by inquiring for any idiosyncrasy against quinin or belladonna before continuous doses of these remedies are prescribed. If the abortive treatment is not successful much may be done for the comfort of the patient by the use of sprays, irrigations, and pigments. The various preparations of the suprarenal gland are useful. Adrenalin chlorid diluted with normal salt solution (1:10,000) with or without cocain or eucain (1 per cent to 2 per cent) affords relief. The nose may be irrigated with a mild solution of Pulv. Antisepticus Co. U. S. P.

The anterior douche must be used with caution. The patient's head is tipped well to the left, and he is requested to take and hold a long breath while the right nostril is slowly irrigated with the mouth open. The head is then tipped to the right and left nostril irrigated in the same manner. Viscid tenacious secretion is dislodged by this method to the great relief of the sufferer. This procedure is free from danger if performed with gentleness and correct technic in properly selected cases. A spray or application should follow of:

Pulv. mentholis.	
Pulv. camphoræ	āā gr. v. (0.3 gm.)
Petrolati liquidi	℥i (32.0 gm.)

A good office application that will reduce and hold the annoying intumescence is cocain 2½ per cent, antipyrin 2½ per cent. If suprarenal extract or adrenalin is used care must be exercised not to introduce these solutions without considerable dilution. Harm is often done by the use of astringent and irritating sprays.

In the subsiding stage a capsule of cinchonidin sulphate, gr. 2 (0.12 gm.), and hydrastin (white alkaloid), gr. ¼ (0.015 gm.), is efficacious. In children the tincture of chlorid of iron with glycerin and water is valuable. If subacute intumescence prevails the application of the galvanocautery affords relief during the winter season. In infants or young children relief may be secured by the instillation of a solution of adrenalin (1:10,000), followed by argyrol or silvol 20 per cent—put 5 to 10 drops into the nostril with a medicine dropper. Inhalations of 1 part menthol to 10 to 20 parts of chloroform may be used on a handkerchief, or with a convenient nose and mouth inhaler.

As acute rhinitis is usually caused by microbic infection, an investigation of the bacteriology will reveal most frequently which are the microorganisms concerned: catarrhalis, staphylococcus, streptococcus, pneumococcus, or influenza bacillus. Great curative value is claimed for the autogenous vaccine made from the culture. When the laboratory equipment is not available a stock vaccine with urotropin internally may be given. The true worth of this treatment must be demonstrated by future clinical reports.

Chronic Rhinitis.—Anatomic or pathologic conditions that are sufficient to interfere with nasal drainage or proper respiration must receive surgical attention. The etiology should be carefully investigated, and systemic dyscrasiæ, such as gout, rheumatism, tuberculosis, syphilis, or focal infection, cardiac and renal insufficiency, must be given appropriate treatment. The excessive use of alcohol, tobacco, or drugs will prevent successful treatment, and they must be discontinued or modified. Habits of personal hygiene should be considered, and details relating to proper clothing, exercise, bathing, and ventilation of the working and sleeping apartments should be subjects for instruction to each individual sufferer. The warm and dry climates of Egypt, Mexico, Arizona, New Mexico, and the Northwest are often advisable when the leisure, the financial condition, and inclination of patients will permit travel and absence from home. Among children with a tendency to overactivity of the lymphatic system the syrup of the iodid of iron or the syrup of hydriodic acid would be an important aid to local treatment. The early removal of adenoids of the nasopharynx, and offending tonsils, offers a valuable prophylactic measure in limiting acute inflammations in the upper respiratory tract, and also of the chronic rhinitis of adult life.

Every case of chronic rhinitis presents two chief therapeutic indications: the establishment of thorough cleanliness by the restoration of free nasal drainage and free nasal respiration. These must be accomplished by the destruction of the least possible amount of mucous membrane. If mild pathologic changes have occurred medicinal methods may be sufficient. Cleansing sprays or douches of Dobell's or Seiler's or of the normal salt solutions; or a solution of potassium permanganate, gr. ⅛ to ℥i, or a solution of Pulv. Antiseptic Comp. ½ teaspoonful to the pint will be sufficient. Many escharotics have been advocated. Those of real power, however, are chromic and monochloracetic or trichloracetic acid. In the intumescent varieties these measures, especially with children, afford prompt relief—in properly selected cases. The fused bead of chromic acid is preferable to many plans, but the danger of excessive cauterization is imminent if the acid runs on the floor of the nose or extends over an uncontrollable area. The tissue should be dried before the acid is applied. Mild intumescent and hyperplastic conditions may be improved by applications every third day of argyrol, 20 to 50 per cent, or nitrate of silver, 5 to 10 per cent, or iodin, grains x, to glycerin ℥ss, aqua ℥ss.

Many favorable results are reported from electrolysis. Five to 10 m.a. are passed through a double needle by the bipolar method for three to five minutes. The galvanocautery affords the best and most definite method (except those strictly surgical) of reducing intumescence and hyperplasia. Free nasal drainage and respiration may be quickly and simply restored in moderately severe and otherwise suitable subjects by this procedure. The inferior turbinates should be cocainized with a 2 to 4 per cent solution, or alypin, 10 per cent, before operation. A deep line should be drawn from the posterior portion of the turbinate body anteriorly, and extending down to the periosteal layer. The wound should be covered with a bland oily spray followed by an insufflation of aristol. The middle turbinate should not be cauterized. When these measures appear to be ineffective (or, in fact, in all cases of marked nasal obstruction) prompt surgical methods should be adopted. The submucous resection or anterior and posterior turbinectomy of the inferior and middle bodies are operations which when indicated afford splendid results.

Membranous Rhinitis.—The plan of therapy adopted in pseudomembranous rhinitis is dependent in part upon the bacteriologic findings. The presence of Klebs-Loeffler bacilli, particularly with a secondary infection elsewhere, demands an early dose of antidiphtheritic serum, the initial strength of which should be 5,000 units. In mild primary infections among adults under continuous observation antitoxin may be held in reserve. As the involvement of the nasal mucous membrane may be transmitted in like form to those under immediate exposure, the prophylactic use of antitoxin may be necessary. The usual dose of calomel, followed by an appropriate saline, such as Rochelle salts, will be indicated. Locally a thorough cleansing treatment should be prescribed. For this purpose the posterior, or carefully directed anterior, douche of warm normal salt solution, boric acid, soda bicarbonate, or liquor calcis, U. S. P., will lessen the absorption and aid the exfoliation of pseudomembrane. Peroxid of hydrogen may be applied directly to the parts with a cotton pledget. It inhibits the bacterial growth. It must be remembered that the separation of the pseudomembrane from the nasal mucosa leaves a raw and frequently bleeding surface; therefore, it should be neither forcibly detached nor treated with powerful antiseptics or astringents. The excoriation may be treated with advantage by applications of silver nitrate [10 to 20 gr. (0.6 gm.) to the ounce (30 c.c.)] or a copper sulphate solution. Aristol insufflated freely is particularly beneficial. When the acute symptoms have subsided tonic treatment such as the elixir of calisaya, iron, and strychnin should be administered. Iron and strychnin may be given in daily hypodermic doses, .05 iron citrate, .001 strychnin sulphate.

Atrophic Rhinitis.—A glance at the various textbooks reveals the fact that the number of remedies advocated for the treatment of "atrophic rhinitis" is quite as great as the inefficiency and uselessness of the majority of them. A definite plan must be selected and continued for some months if good results are to be expected. The treatment should be general and local. Attention must be given to hygienic details, and all exposure avoided when possible. An even, warm, moist climate favors recovery, and many patients improve rapidly in a subtropical climate. The use of the iodids of iron, arsenic, or potassium is indicated, and their tonic and alterative action on the glandular apparatus is beneficial. Hypodermic injections of the citrate of iron and strychnin are very effectual in anemic cases. The hemoglobin may be raised 5 per cent a month by this method. A comparatively painless hypodermic injection of iodin, 10 per cent (iodolena), in almond oil may be injected, intramuscularly when the stomach is irritable or easily disturbed by the iodin salts.

If the atrophic process is attended by thyroid deficiency, thyroid proteid extract or iodin (gr. 1/12) prepared in cubes of gelatin (gr. 1/12 to each cube—1 by ⅛ inch) may be given three times a day with milk, before eating, in addition to iodin by electrophoresis. A general tonic effect on the glandular system may be produced by the soap bath. The patient strips in a warm room and thoroughly covers the body with a thick lather of white castile soap, the smallest quantity of water being used. A flesh brush is now quickly applied over the whole surface of the skin for a period of five minutes, when the redundant lather is removed with a Turkish towel and sharp friction. The patient then returns to bed. Tonics of strychnin, iron, or phosphorus cap. gr. 1/100 in oil should be continued throughout the treatment.

Thorough local cleansing measures are essential. A plan of office and of home treatment should be chosen which shall involve simplicity, economy of time, expense, and effort. The methods adopted must be carried out through a long period of weeks or months. Results are obtained only by the exercise of patience and persistence. Especially with children it is necessary to use alkaline solutions that are pleasant in taste and odor for cleansing purposes. Of the official preparations the solutions made from Seiler's tablets, sodium chlorid, borax, or boric acid are convenient, cheap, and efficient. Innumerable preparations have been marketed that contain these essential ingredients, whose color and odor are made attractive. The efficiency and value of them, however, depend upon their alkalinity and mild antiseptic properties. In severe cases, where the crusts are dry and tenacious and show marked decomposition, potassium permanganate, 1/12 gr. to the ounce, peroxid of hydrogen, or sol. mercuric chlorid (diluted 1 : 7), 1 : 3,000, may prove of greater usefulness than the above-mentioned preparations. These solutions should be warmed and either sprayed or syringed through the nasal passages both anteriorly and posteriorly. J. C. Teal reports pronounced results from the use of water soluble scarlet red (Biebrich ozo-benzin). After an alkaline douche or spray a 4 per cent scarlet red emulsion is applied to all parts of the nasal mucous membrane with gentle massage. The application is renewed every forty-eight hours, although the odor and crusts may be absent after the second application.

If these measures are insufficient to remove crusts and infected secretions from the nasal channels, cotton swabs saturated with peroxid of hydrogen should be used. Treatment often fails because thorough cleansing methods have not been adopted. The nasal passages should be carefully examined after the douche or sprays are made, in order to determine satisfactorily the condition of the nasal cavities before a routine treatment is instituted.

Of the various oleaginous preparations for applications or sprays that have been recommended in great number, oleum picis and balsam of Peru may be considered among the best and safest. Thymol, menthol, and camphor may be used from time to time as adjuvants and as convenient preparations for home treatment, however. G. L. Richards favors a douche containing 5 to 10 drops of formaldehyd in 8 ounces of warm water, preceded by an application of cocain. In the more advanced stages of atrophy the milder methods are inefficient and additional stimulating treatment must be chosen. No cases that afflict these unfortunates call for greater mutual courage and persistence on the part of the patient and physician than the chronic atrophic forms that have developed ozena with all its depressing conditions. Galvanism and faradism applied over a period of months may give well-earned results. The current should be passed through a naked electrode with a ball-shaped distal extremity. The electrode should be long and slender and all atrophic regions should be reached if possible. The treatment is commenced with 1 to 5 m.a. and gradually increased at each successive sitting. The cathode is placed against the auricular fossæ while the anode is passed rapidly over the atrophic areas. Later the faradic may be substituted for the galvanic current. The application is usually followed by a reactionary hyperemia, and if any excessive irritation follows it may be allayed with a spray or application of menthol in petrolina oil. The application may be repeated daily at first and gradually reduced to twice a week. Massage is a favorite method with many laryngologists. Instruments for this may be applied by hand, the electric motor, or a special vibratory apparatus. Kelligrew claims to have treated over a thousand cases with marked improvement by vibratory and light stroking movements applied by means of a sound 20 cm. in length. The instrument is covered with a rubber head over some cotton. The floor of the nose, meati, inferior and middle turbinate bodies, and septum are treated in order. W. Freudenthal has designed a good instrument for employing electrical vibratory massage.

The tampon suggested by Gottstein has been recommended extensively. This may be placed by means of a screw applicator into one passage and allowed to remain four or five hours, when the other passage is treated in a like manner. Various modifications have been devised by Percher, Hubbard, and Leland, whereby pigments of ichthyol, iodin, or an alcoholic solution of acetanilid are applied by this method. This plan is frequently annoying and obnoxious to the patient, and may fail for this reason.

The most efficient topical application is: iodin, gr. vii to xii; glycerin, ℥ss; aquæ, ℥ss.

Ichthyol, 20 per cent, argyrol, 20 per cent, or resorcin, followed by a spray of menthol and camphor, 5 gr. each to the ounce of petrolina oil are valuable. Special investigation by X-ray and illumination should be made to determine the condition of the accessory sinuses. Proper surgical measures and ventilation often afford great relief.

RHINOSCLEROMA

Rhinoscleroma is exceedingly rare in this country. Only 3 cases were reported in the United States before 1893. The pathology and treatment of this disease have been most carefully studied by Emil Mayer and Gertser of New York, and by Chiari, Ganghofner, and Paultauf of the Old World. Freudenthal has suggested the value of Coley's fluid or rhinosclerene proposed by Pawlowsky. The use of autogenous vaccine has apparently offered something of inhibitory value in a few cases. Five cases have come under the writer's observation with laryngeal involvement that were relieved for a time by dilatation with Schroetter's tubes. In Lubliner's case the lesions disappeared after an attack of typhoid fever. Mercury and iodids have been used extensively without success. Temporary relief has been accomplished with the various chemical caustics. Palliative surgical methods are recommended in selected cases to relieve stenosis.

SINUSITIS

Acute Nasal Sinusitis.—Acute inflammation of the mucous membrane lining the nasal accessory sinuses is a common complication of acute nasal catarrh. In some of the grip epidemics involvement of the nasal accessory sinuses is extremely common. The inflammatory process may be restricted to the sinuses on one side, affecting one or several simultaneously, or it may involve all the sinuses on both sides, producing a condition of pansinusitis. An acute abscess about the root of an upper molar or even a bicuspid tooth not infrequently ruptures into the antrum and produces an acute maxillary sinusitis. Acute sinusitis is always associated with more or less marked subjective symptoms, which vary from a sensation of fullness or stuffiness on the affected side to that of pressure or even severe pain over the region of the sinus involved, that is, over the frontal or maxillary sinuses or between the eyes. The recognition of an acute sinus inflammation presents no great difficulty. The process can usually be suspected from the subjective symptoms complained of. In addition to the sensations over the region of the involved sinus and the blocking of the nasal passage on the affected side, there is usually present a marked increase in the nasal discharge. The transillumination test will usually give a positive finding for both the maxillary and frontal sinuses, easily recognized when the process is unilateral (see discussion of Transillumination Tests under Chronic Sinusitis). The skiagraph will assist in making the diagnosis, especially in cases of frontal sinus trouble. Should the skiagraph leave any doubt concerning the maxillary sinus, this can readily be cleared up by irrigation of the sinus, by introducing a cannula into the natural opening, or by puncturing the nasal wall with a suitable instrument either in the middle or the inferior meatus.

The treatment of acute sinusitis is chiefly directed to the relief of the subjective sensations. If the maxillary sinus has become infected by a tooth abscess, it is important first of all to have this abscess taken care of by a dentist. The symptoms of nasal obstruction are relieved by reducing the turgescence of the nasal mucous membrane. The simplest means for relieving this condition is by nasal irrigation, using hot normal salt solution. In order to avoid the risk of carrying the infection up the eustachian tube into the tympanum, it is best to direct the patient to draw the salt water up into the nose. This can be facilitated often by the use of a straight glass tube with a suitable bulb on the nasal end. A great deal of relief is usually afforded by spraying the nose with the following preparation: menthol, gr. 8; camphor, gr. 4; oil of eucalyptus, minims 3; albolene, oz. 2. The patient should be directed to draw the albolene spray back through the nose. A weak solution of adrenalin, 1:10,000, sprayed into the nose is often an effective way of relieving the intranasal swelling. The most effective method for relieving the intranasal obstruction is by applying with a cotton swab a ½ per cent solution of cocain in 1 : 10,000 adrenalin along the under surface of the middle turbinated body. The subjective sensations of pressure and of pain result from the retention of secretion in those sinuses which have their openings under the middle turbinated body. This includes the maxillary and frontal sinuses, as well as the anterior ethmoid cells. The relief afforded by applications along the free edge of the middle turbinated body lasts usually for several hours and in some cases for the larger part of the day. Suction by means of a suitable apparatus may be employed to withdraw secretion from the accessory cavities in cases of acute sinusitis. The value of this method of treatment is secondary to that outlined above. The use of the electric light head bath devised by Brünings is usually an effective method of relieving intranasal turgescence and the subjective symptoms dependent on this condition. Severer cases should be kept in bed. Aspirin, 5 gr., repeated every three or four hours, often affords relief. In other cases, an opiate may be necessary.

The necessity for treatment further than that just outlined for the relief of acute sinusitis arises only in the exceptional case. One occasionally encounters a case where, because of the failure to secure proper drainage through the normal openings by the methods already given, the pain occasioned by the retention of secretions becomes so severe that resort must be had to surgical measures. In the case of the maxillary sinus the cavity can be irrigated by puncturing the nasal wall of the sinus either in the middle or the inferior meatus. A few irrigations usually suffice to clear up the process. Should the severe symptoms persist, a large opening can be quickly made through the nasal fontanel in the middle meatus.

For the relief of retention in the frontal sinus and in the anterior ethmoid cells a resection of the anterior end of the middle turbinated body will usually suffice. In exceptional cases this may have to be followed by an exenteration of the anterior ethmoid cells. Irrigation of the frontal sinus by the introduction of a suitable cannula will relieve the pain occasioned by the retention in this sinus. There are very few cases where all efforts to secure intranasal drainage for an acute frontal sinusitis fail. In these cases one may be forced to open the sinus by an external operation in order to relieve persistent severe pain.

Chronic Nasal Sinusitis.—Chronic inflammation of the nasal accessory sinuses appears under several distinct forms. The trouble may be limited to one sinus or there may be involvement of all the sinuses even on both sides of the nose. Chronic foci of infection in the nasal sinuses may assume an important part in the etiology of systemic infection. The close relation of the sinuses to the orbit permits of a direct extension involving the eye, a condition which is particularly apt to occur from disease of the ethmoid. The anatomical relation which frequently exists between the optic nerve and the posterior ethmoid cells and sphenoid sinus permits of an involvement of this nerve by an extension of the infection from these sinuses. The profuse discharge of pus in some cases of chronic sinusitis draining back into the pharynx may cause digestive disturbances. The detection, therefore, of cases of chronic sinusitis and the institution of the proper method for relieving this condition becomes a very important question.

The symptoms complained of by the patient are not so characteristic as in cases of acute sinusitis. The persistence of a profuse purulent discharge from the nose should always arouse suspicion of a chronic accessory sinus disease. A unilateral discharge is particularly characteristic of sinus empyema. In a good many cases, the process is more or less latent and the trouble can be detected only after a careful search. In cases where the amount of discharge is slight it will sometimes form in crusts about the nasal orifice of the affected sinus. Headache is characteristic of chronic sinusitis, especially during periods when there is some obstruction to the escape of the secretion from the sinus, or during the period of an acute exacerbation of the chronic process.

The method of procedure by which the detection of chronic sinusitis is made consists in the first place of an intranasal examination by means of reflected light. Purulent secretion found in the nose should arouse suspicion of chronic sinusitis. The reappearance of pus in the course of a few minutes after the nasal passages have been carefully cleansed is proof that the secretion comes from some reservoir where it has been previously accumulating, that is, from an accessory sinus. If the pus reappears under the middle turbinated body, that is, in the middle meatus, it means an involvement of the maxillary sinus or frontal sinus or of the anterior ethmoid cells, or of all three. If the pus reappears between the middle turbinated body and the septum, it can come only from the sphenoid sinus or the posterior ethmoid cells. In a great many cases of chronic sinusitis, the amount of secretion is so slight, especially during the latent stages, that little or no secretion is found in the nose, and none reappears in a reasonable period after cleansing. The second step in the examination is to apply the transillumination test. Should this show a distinct shadow over one maxillary sinus while the other remains clear, it should arouse strong suspicion that the sinus is involved, and in case the shadow is on the side where pus was found to reappear in the middle meatus, there is little room to doubt the presence of maxillary sinusitis. Not infrequently when the bones are heavy, transillumination leaves one in doubt, because of the presence of a shadow over both sinuses. When, on the other hand, transillumination shows the absence of a shadow on either side, we are fairly safe in concluding that there is no maxillary sinus trouble. The transillumination of the frontal sinus is not as a rule conclusive because of the frequent occurrence of small sinuses which fail to give a positive difference on the two sides even when only one sinus is affected. In many cases, especially where the sinuses are not small, the transillumination will be found so clear as to preclude the possibility of frontal sinus disease. In other cases, too, when the sinuses are not small, the presence of a distinct shadow over one sinus, when contrasted with the clear illumination of the opposite side, leaves little room to doubt the presence of a frontal sinus empyema, especially when this finding is combined with the reappearance of pus in the middle meatus.

In the cases where the intranasal examination and the transillumination tests do not exclude the possibility of sinus disease, a skiagraph should be made. This will be of value in determining not only the presence of trouble, particularly in the case of the frontal sinus, but the skiagraph will also show the exact size of the frontal sinus. The final step in making a positive diagnosis in suspected cases is by irrigation of the sinus in question. This method has its greatest use in cases of maxillary sinus infection. The irrigation is accomplished by catheterizing the normal opening in the middle meatus or by puncturing the nasal wall of the sinus in the middle or the inferior meatus. If pus is washed from this sinus and reappears again in the middle meatus it must come from either the frontal sinus or anterior ethmoid, or from both. Irrigation of the frontal sinus is not so simple and cannot always be accomplished, even after the anterior end of the middle turbinated body has been removed. If pus reappears in the middle meatus after irrigation of both maxillary and frontal sinuses, it can come only from the anterior ethmoid cells. Irrigation of the sphenoid sinus is more readily carried out.

This preliminary discussion of the detection of chronic sinusitis will help to clarify the problems one encounters in relieving these conditions. Not infrequently a tooth abscess has been the starting point for a chronic maxillary sinusitis, which in turn has infected the other sinuses on that side. It is important in such cases that the offending tooth be properly taken care of first of all. The primary factor in the successful treatment of most cases of chronic sinus disease is to secure adequate drainage and ventilation of the sinus. This accomplished, the condition often tends to heal without further treatment. In the case of the maxillary sinus, this drainage can be accomplished by making a sufficiently large opening either in the middle meatus in the region of the nasal fontanel, where the most fragile part of the nasal wall of the sinus is found, or under the inferior turbinated body. A large permanent opening made in this way is usually sufficient to bring about a cure of chronic maxillary sinusitis. The operation may be followed by daily irrigation with a warm normal salt solution through the opening into the sinus. This the patient can be taught to carry out by himself for a few weeks until the pathologic secretion has disappeared. In intractable cases a more radical operation may be called for. This consists of making an incision under the upper lip and lifting the periosteum over the anterior wall of the sinus. This wall is then removed and a greatly thickened mucous membrane in the sinus curetted away. The operation is completed by making a large permanent opening into the inferior meatus of the nose.

The cure of chronic frontal sinusitis is undertaken in the same way by securing adequate drainage into the nose. Since the frontal sinus disease is usually complicated by a more or less extensive involvement of the anterior ethmoid cells, the first step in securing drainage for the frontal sinus is the removal of the anterior part of the concha media, followed by a partial or a complete exenteration of the ethmoid cells, depending on the extent to which they appear to be involved. Finally an effort is made to increase the size of the nasofrontal opening. Considering the relation of the ethmoid cells to the orbit and to the cribriform plate it is evident that operations in this region should be undertaken only by those who have made a most careful study of the regional anatomy of this part, and are also perfectly familiar with the technic of intranasal operations. This operation for securing better drainage from the frontal sinus may be followed by regular daily irrigation of the sinus with a warm normal salt solution. This too, as a rule, the patient can acquire the ability to carry out at home. Many cases of chronic frontal sinusitis go on to a perfect recovery after this treatment has been carried out. In other cases the trouble persists because of permanent alterations in the membrane lining the sinus, usually in the form of polypoid degenerations, which are sometimes found completely filling the cavity of the sinus. The disfigurement, more or less marked, which is certain to result from the scar of an external operation on the frontal sinus is a contra-indication to this procedure, except in unusual cases where, after every effort has been made to secure relief by drainage through the intranasal route, the severe pain continues or symptoms of an intracranial extension appear.

The radical treatment of chronic empyema of the ethmoid labyrinth or of the sphenoid sinus is much simpler. This consists in the exenteration of the ethmoid cells and the resection of the anterior wall of the sphenoid sinuses. Both of these operations are now feasible and are carried out intranasally by the specialist skilled in the technic of this sort of work. Sometimes it is not feasible to accomplish the complete exenteration of the ethmoid labyrinth at one sitting, and in these cases repeated sittings require a great deal of perseverance both on the part of the patient and of the surgeon to search out and drain all infected cells. Even a small concealed pocket of pus in such cases may constitute the focus for the persistence of a most serious systemic infection.

The results obtained in the treatment of chronic nasal sinusitis may be summed up as follows. By securing adequate drainage by intranasal operations most cases can be permanently cured. The sinus most likely to persist in discharging pus in spite of this treatment is the frontal sinus, and here the advisability of an external operation is particularly contra-indicated because of the conspicuous scar which must result. In those cases where by intranasal surgery we fail to bring about a definite cure of frontal sinusitis, this treatment affords in most instances relief from the chief symptoms complained of, namely, frontal pain, occasioned by the obstruction to the outflow of pus from the sinus. The complete elimination of foci of infection from remote ethmoid cells presents great difficulties and these may be met with when an external operation is undertaken as often as when one relies upon intranasal surgery. The complete eradication of chronic foci of infection, therefore, in cases where these are suspected of causing systemic disease may not always be entirely feasible, especially where such foci are located in remote ethmoid cells or in the frontal sinus.

Hyperplastic Ethmoiditis.—In connection with chronic accessory sinus disease mention should be made of the condition of polypoid degeneration of the mucous membrane lining the ethmoid cells, as well as of the membrane about the nasal orifice of these cells, especially of that covering the uncinate process in the middle meatus. This form of ethmoiditis is not primarily a suppurative disease. It is not uncommon, however, for a subsequent infection of the ethmoid, such as frequently occurs from an attack of influenza, to produce a suppurative condition which, because of the impaired drainage occasioned by the presence of polypi, is very prone to become chronic. This condition of polypoid degeneration of the ethmoid seems often to be predisposed by the presence of an anatomical variation whereby the normal ventilation and drainage of the ethmoid cells are readily impaired. A large percentage of the cases occur in patients where the concha media is wedged so tightly between the septum and the outer nasal wall as to practically close the middle meatus, into which the chief ethmoid cells open. The anatomical relation is such that but a slight congestion of the mucosa of the nose is required to close tightly the openings of the cells. The symptoms observed in these cases, particularly in the early stages, are chiefly attacks of sneezing associated with a profuse watery nasal discharge. There may be at times a sensation of fullness or pressure between the eyes. The voice often lacks its normal resonance, just as occurs frequently in acute coryza. Sooner or later in a great many of the cases distinct symptoms of asthma develop. Indeed, it is rather the exceptional case of asthma where distinct evidence of a hyperplastic ethmoiditis cannot be detected. A casual examination of the nose very often fails to discover this condition, for the reason that an inspection of the middle meatus is interfered with because of the anatomical condition described above and by the time the polypi begin to make their appearance from under the concha media the change in the ethmoid cells is already of long standing. In such cases, where because of the symptoms one is led to suspect a hyperplastic ethmoiditis, it is necessary, by means of a long-bladed nasal speculum (a Killian speculum), to pry open the middle meatus in order to detect evidence of beginning polypoid degeneration. The condition is, of course, a chronic one and there is no tendency for a spontaneous recovery. In the early stages the improved ventilation of the ethmoid cells, accomplished by the resection of the anterior half of the concha media, seems often to be sufficient to bring about a cure. In the later stages only the complete exenteration of the ethmoid labyrinth by a radical intranasal operation will be of permanent value. These operations bring relief more frequently of the strictly nasal symptoms than they do of the associated asthma. Failure to cure the asthma in some cases may depend upon our inability to accomplish such a complete exenteration of the ethmoid cells as to prevent the recurrence of polypi.

EPISTAXIS

In the treatment of epistaxis, or "nosebleed," it is of the utmost importance to determine the causal factors. A careful examination with a good light and nasal speculum should be made. The traumatic varieties will call for topical applications. The simple domestic methods of relief, such as holding the hands above the head, the application of ice to the alæ and root of the nose, may be sufficient. As the majority of simple cases come from a rupture of the septal artery at Kesselbach's spot, a short distance within the vestibule, a cotton tampon inserted and resting over this area, with pressure from the outside, will promptly arrest an ordinary hemorrhage. The tampon may be saturated with adrenalin chlorid (1:1,000), or cocain and antipyrin (each 2½ to 5 per cent), by peroxid of hydrogen or thromboplastin. In every case that is not relieved by these simple methods the bleeding point should be found and treated. The habits and constitutional dyscrasia of the patient must be carefully studied.

In atheromatous subjects, or those with high blood-pressure, epistaxis is often a safety valve of nature that may prevent impending disaster in a more remote region of the body. When the bleeding from the septal artery or from granulations on the turbinate bodies is excessive or recurrent the application of trichloracetic acid, the nitrate of silver stick, or the galvanocautery point (applied and removed at red heat) will give prompt results. When the ethmoidal arteries are bleeding, a bismuth or iodoform gauze tampon to the roof of the nose will prove successful.

If the anterior method of nasal tampon is chosen it is essential that either dressing or nasal forceps with fine blades should be used, and strips of gauze, ½ inch in width, passed well back into the posterior portion of the nasal canal, followed by others until firm and uniform pressure is effected. The gauze may be saturated with suprarenal extract, or dusted, if necessary, with tannogallic acid powder. The use of Monsel's solution, chlorid of iron, or other powerful astringents is a method of last resort, but objectionable on account of forming hard clots.

In severe postoperative epistaxis, or in persistent hemorrhage after the simple methods have failed, it may be necessary to use a posterior tampon. This is prepared from cotton or gauze, by trimming and rolling a suitable piece to a size that will enter the posterior opening and not easily pass forward into the nasal passage. It should measure for an adult about 1¼ by ⅞ inches. A string should be passed around the middle of the tampon, leaving two long ends. A small soft catheter should be passed through the bleeding side into the pharynx, where it is seized with a long forceps, and the cord drawn out of the mouth cavity. One of the free ends of the string is tied firmly to the catheter and the latter withdrawn through the nasal passage, while the tampon is forced gently into the posterior opening by the index finger of the free hand. The ends of the strings from the nose and mouth are then fixed with adhesive plaster.

Internal medication is especially indicated in cases of renal or cardiac (vascular) disease attended by high blood-pressure. This may be accomplished by the use of tincture of aconite in drop doses of 3 to 5 drops at once. Nitroglycerin (gr. 1/100) hypodermically will act promptly, or this preparation may be given every half hour for three or four doses. If the blood-pressure is low, tincture of digitalis (minims 5) every hour for three doses, or camphor, or ammonia, will be more beneficial. When the hemorrhage is continuous or profuse, hydrastinin hydrochlorid (gr. ¼) or stypticin (gr. 1/3) hypodermically may be efficacious. In persistent cases due to hemophilia, anemias, or allied disorders the hypodermic use of antitoxin or freshly prepared horse serum is of value. Calcium lactate in 15-gr. doses hourly, by raising the coagulation point, may be necessary. Gelatin in large quantity has a similar influence. The use of ergot is unscientific and harmful from the fact that valuable time is lost. Therefore a more suitable remedy must be chosen. In recurrent and persistent epistaxis the presence of postnasal ulceration or a tumor, or the possibility of pulmonary hemorrhage, must be eliminated.

OZENA

While comparatively few cases of recovery from ozena are reported, yet in the majority of cases it is possible to outline a plan of treatment that will afford great comfort and temporary relief. This plan should always begin with thorough cleansing of the parts. It is important to improve the general physical condition and to prevent the chemical changes which unhealthy secretions undergo. It is only by the utmost diligence that satisfactory results may be obtained.

The patient should be instructed in the use of the antiseptic alkaline solutions such as Seiler's and Dobell's solutions, or compound antiseptic powder (U. S. P.). It is often necessary to soften the crusts with vaselin or liquid petroleum before the cleansing wash is used. This routine on the part of the patient should be followed by efforts to prevent effective crust formation and its decomposition. Internal administration of the iodid of iron, cod-liver oil, phosphorus, strychnin, or arsenic will be frequently indicated. Plenty of outdoor life and exercise with good nourishing food should be ordered (see Atrophic Rhinitis).

The special treatment of this disease offers many different plans and agents under the theory of the infective character of ozena. The hypodermic use of antidiphtheritic serum has been proposed and highly recommended by Gradenigo. The duration of treatment varies from thirty to sixty-eight days, during which time 5,000 to 48,000 units of serum were employed. Perhaps the most efficacious local application is a 2 per cent solution of creolin followed by ichthyol, 20 to 50 per cent, and a mixture of iodin and oil of cloves. Massage and electricity are beneficial methods of treatment. In some cases the stimulating effect of iodin, gr. 48; potassium iodid, 16 gr.; water, 1 ounce; glycerin, 1 ounce; is followed by improvement.

The hypodermic injection of iodin is highly recommended. This may be given in the 5 to 10 to 20 per cent preparations in almond oil (iodelaine). The stronger preparations are more or less painful. A spray of bichlorid of mercury, 1:3,000, is an excellent medicament in cases that are possibly luetic in origin. Remarkable results have been claimed by Horn of San Francisco from the use of a vaccine prepared from the isolated infecting microörganism which he claims is the etiologic factor. Thorough surgical attention to the pathology of the sinuses must not be neglected.

CHAPTER II

DISEASES OF THE LARYNX

Burt R. Shurly

LARYNGITIS

Acute Laryngitis—*Prophylactic and Internal Treatment.*—The prophylactic and internal treatment of acute laryngitis, or acute laryngeal catarrh, has much in common with acute inflammation in the nasal passages, nasopharynx, and pharynx. The use of aspirin and Dover's powder (in 3 to 5 gr. dosage) is beneficial and comforting. The use of the voice should be forbidden, as rest is an essential measure, especially at the outset or early stage. Advice in this regard must be firm, as rest will decidedly favor other abortive measures. The upper respiratory cavities should be examined, as acute laryngitis is rarely the primary affection, nevertheless the attending acute inflammation should receive equal attention. Small and frequent doses of tincture of aconite root, 1 to 2 minims every hour until the constitutional effect is noted, will often relieve local congestion and the pyrexia. If pain be prominent aspirin often gives prompt relief. It may be administered in rheumatic subjects together with salicin. In malarial districts the addition of quinin is advantageous. The early use of calomel in 1/10-gr. doses until ten or fifteen are taken is advised. This should be followed by the usual saline in the morning. The cold compress over the larynx, covered with an oiled silk pad, is grateful to many patients. The ice collar or Leiter coil may be adopted if convenient. However, the danger of additional congestion of the parts after continued hot applications makes them of doubtful utility. A room saturated with moist soda steam is grateful and comforting. A croup tent may be improvised and supplied with alkaline steam (soda bicarbonate, 1 ounce to a quart of water) from a kettle to which a piece of garden hose has been attached to the spout. Various steam inhalers are in the market. A pitcher of boiling water with a cornucopia of paper placed over it as an inhaler affords a satisfactory method of supplying a medicated vapor. A favorite and useful formula is: menthol, gr. ii; tinct. benzoin comp., ʒss; tinct. opii comp., ʒss. A teaspoonful of the mixture is added to a pint of boiling water and placed in the inhaler or pitcher. The medicated steam is inhaled as hot as possible for five to ten minutes. This may be repeated at hourly intervals. Oil of eucalyptus in the proportion of 1 ounce to the quart of water may be used in the same manner or boiled over a spirit lamp.

Oleum terebinthinæ, oleum pini, and oleum juniperi are also to be recommended, to be used in the same way. When the larynx is dry and a tickling sensation with a short frequent cough is present, then tartar emetic in careful dosage is indicated. This may be administered pleasantly to adults in the following prescription:

℞	Antimonii et potassii tartratis	gr. ss
	Tincturæ ipecacuanhæ et opii	ʒss
	Liquoris potassii citratis	℥ii

Sig. ʒi every two hours.

Some of the various lozenges that contain ammonium chlorid or menthol and camphor are very acceptable. In very young children a powder containing ½ to 1 gr. of cinchonin with 1/12 gr. of ipecac will often give great relief. The cough in adults may be relieved with heroin gr. 1/12 to 1/24.

Local Treatment.—The upper passages should be cleansed with a spray or irrigation of warm normal salt solution. Dobell's or the Seiler solution may be preferred. An attempt should be made by every practitioner to eliminate from use a vast list of proprietary preparations which are to be found on the shelves of the laity, and which are used to the detriment, many times, of the delicate nasal mucous membrane of the patient. The therapeutic properties of these solutions are generally based upon the ingredients of the original solutions of either Seiler or Dobell. Many of these proprietary solutions are compounded of inferior materials and of a strength (of solution) that is not only harmful but frequently permanently injurious. The choice in the U. S. P. of salt, Dobell's or Seiler's solution, Pulv. Antiseptic Comp., etc., is amply sufficient to fulfill all indications.

An inhalation spray of menthol and camphor, gr. ii to the ounce, in albolene is valuable and soothing, especially in the dry stage of the attack. To use the spray effectually the anterior portion of the tongue is to be wrapped with a piece of gauze, then extended and held by the patient, while the alæ of the nose are compressed by the operator to prevent nasal respiration. The patient now inhales and exhales while the spray is continued. By this method the mucous membrane of the larynx is more quickly and thoroughly treated. A down spray, consisting of alum (gr. x), acid carbolic (gr. ii), glycerin (½ ounce), to ½ ounce of water, may be applied directly to the larynx with benefit. Strong astringents should be used only exceptionally. Temporary relief may be obtained by a laryngeal spray of adrenalin 1 : 10,000.

In children, in whom edema develops frequently, a dose of turpeth mineral or syrup ipecac, used as an emetic, together with a prompt steam inhalation, will afford suitable means of relief.[1]

Intralaryngeal applications are of benefit in the subacute stage, as argyrol, 20 to 40 per cent, or alumnol, 20 per cent. Spasms of the glottis may prove annoying, and dangerous in some cases. Anodynes such as heroin, codein, or opium, by mouth or hypodermically, will promptly allay the nervous phenomena in such cases. Intubation, or even tracheotomy, may be necessary in rare occasions.

It is important to adopt every therapeutic measure to maintain and preserve the general good condition of the patient. Reconstructive or nerve tonics, such as nux vomica and strychnin, with quinin and iron, are frequently indicated. The use of alcohol and tobacco should be prohibited during an attack.

Chronic Laryngitis.—This condition may appear as a hypertrophic, a subglottic, or a pachydermatous variety. The hypertrophic form may exist with chorditis nodosa, familiarly known as "singer's nodes," and usually located at the junction of the anterior with the middle third of the vocal cords. The various forms of systemic disease and focal infection must be investigated and treated accordingly. The value of a warm, dry climate must be considered, but recommendations must be based upon the financial and general condition of the patient, the age, and possibilities of travel, etc. Conditions causing nasal stenosis and pathologic areas of hyperplastic gland tissue must be surgically removed. Attacks of indigestion, gout, rheumatism and syphilis should be modified by proper systemic treatment and the removal of foci of infection. In cases that develop nodes a six months' rest is necessary. The use of alcohol and tobacco should be forbidden. Internally, potassium iodid, 10 to 15 gr. (0.6 to 1.0 gm.) t. i. d. may be given with caution to assist in the absorption of hyperplasia. Locally, the use of caustics or astringents is frequently indicated. The former should be applied by the skilled specialist of experience only. The fused nitrate of silver or the galvanocautery may be the methods of greatest usefulness. Locally, laryngeal sprays of alumnol, alum, or zinc, preceded by a spray of Seiler's solution, aid in giving comfort to the patient. They are of no curative value. The same may be said for oil sprays, such as menthol and camphor in albolene. Laryngeal applications should be made with great care and only with a reliable instrument, as the Mackenzie, that will hold firmly the proper-sized cotton pledget. The excess of solution must be carefully removed. Increasing strength of zinc chlorid, gr. x to l (0.6 to 3.0 gm.); nitrate of silver, gr. v to xxx (0.3 to 2.0 gm.); argyrol, 10 to 50 per cent, may be introduced into the larynx with benefit. Laryngitis resulting from recurrent infection should have proper surgical treatment for adenoids, tonsils or accessory sinuses, according to the foci of infection present.

---

[1] In most cases in children the administration of bromid of potassium is promptly followed by relief in spasmodic croup, whether due to laryngitis or a purely nervous mechanism.—Editor.

Edematous Laryngitis.—Edema of the larynx requires active treatment by a brisk cathartic of calomel followed by magnesium sulphate or elaterium, gr. 1/6. Alkaline steam inhalations, hot astringent gargles, a mustard counterirritation over the larynx, followed by ice-bags to the neck, should be ordered at once. If the patient is syphilitic and potassium iodid has been administered it should be discontinued immediately, as this drug greatly aggravates the condition of some patients. A spray of adrenalin (1:10,000) through the nose and into the larynx may temporarily relieve. Its use is attended, however, with the danger of reactionary swelling.

When edema follows the entrance of a foreign body, or after bronchoscopy or the removal of an intubation tube, the use of codein or Dover's powder to allay the restlessness may be valuable. Pilocarpin is favored by some, but its depressant effects should be considered.

This condition is usually associated with systemic disease, such as syphilis, tuberculosis, myxedema, carcinoma, bulbar paralysis, or cardiac, renal, and hepatic disorder, and appropriate diagnosis and general therapy are required. If the edema does not respond to the simpler measures and cyanosis intervenes, scarification of the affected region of the larynx should be undertaken. A 20 per cent solution of cocain should be sprayed or applied over and into the larynx and the excess swallowed. Scarification should be performed under the direction of a laryngeal mirror and reflected light. A laryngeal knife is chosen preferably for the purpose. Ichthyol (30 per cent) may then be applied. The resulting hemorrhage and transudation are usually beneficial. If the dyspnea continues and urgent symptoms develop, scarification could be repeated with a guarded bistoury under direction of the index finger.

In great emergency intubation or tracheotomy must be performed. The former operation is often difficult. If one or two trials are not successful tracheotomy should be performed without delay.

Spasmodic Laryngitis.—False croup, laryngismus stridulus, or spasmodic croup requires prompt differential diagnosis. In children, subject to attacks, adenoids, hypertrophied tonsils and elongated uvula may require operation. A cold sponge over the chest, neck, and region of the larynx may be valuable. Gastro-intestinal disturbances should be avoided by carefully selected diet. During an attack treatment directed toward relaxation of muscular spasm must be administered. A hot bath and a teaspoonful dose of syrup of ipecac are favorite and efficient household remedies. Wine of antimony (15 drops) or antimony and ipecac (āā gr. 1/100) every fifteen minutes until emesis results may be given a two-year-old child. Turpeth mineral (gr. ii) was formerly used by many practitioners, but two fatal cases reported by McPhedran proved the danger possible from its use. Antimony may produce depression, and not more than three doses should be given.

Alkaline steam inhalation is useful from a croup kettle or improvised apparatus. Tincture benzoin compound with paregoric (equal parts), a teaspoonful to a pint of hot water, offers a soothing inhalation. Poultices, stupes, or antiphlogistic preparations are beneficial, but the child must remain indoors for several days if they are used.

The attack is liable to recur on succeeding nights unless an antispasmodic or sedative, such as Dover's powder or antipyrin, is given. The administration of the bromid of sodium or of potassium may render all other medication unnecessary, except local applications. Plenty of fresh air should be allowed, unless the atmosphere is damp and cold. Calomel and milk of magnesia may be prescribed with advantage the day following an attack, or an enema of soapsuds and water may be ordered at the first visit. If nutrition and vitality are lowered, careful regulation of the diet and tonic treatment are recommended. In adults when the spasmodic attack is chiefly nervous a hypodermic of morphin (gr. $\frac{1}{6}$) will afford prompt relief. This medication should not be repeated, and it is unwise to inform the patient in regard to the drug or dosage used.

Membranous Laryngitis.—It has been established after much discussion and experience that membranous croup must be considered (and treated) as a Klebs-Loeffler infection. Therefore antitoxin in full dosage (5,000 units) must be given at the earliest possible moment. This axiomatic practice is so important that no time should be lost in administering this specific, even if a doubt of the diagnosis exists in the mind of the practitioner. We know that death occurs rapidly from mechanical obstruction of the larynx and not from absorption, and the greatest haste is imperative, especially in children of the so-called croupy age. Treatment should be initiated with a brisk calomel purge followed by milk of magnesia. A culture will give valuable information in regard to the variety of microörganisms present. This culture may be taken quickly from the epiglottis and arytenoid region by bending the platinum loop or cotton carrier to a proper angle. It is unwise to use strong emetics in this form of croup, as the depressant effect is often marked, and the stomach becomes quickly deranged. Cold compresses, or better, the ice collar, will be found agreeable and serviceable throughout the attack. The inhalation, almost continuously, of steam from alkaline water is indicated from the onset. An albolene spray containing menthol is soothing to the larynx and allays the painful and dry condition of the membrane. Calomel sublimation may be advantageously used. Ten to 15 gr. (0.6 to 1.0 gm.) of the pure powder may be incinerated slowly on a hot shovel or on a piece of metal held over an alcohol lamp. Inhalation is facilitated by constructing a croup tent with an open umbrella and a sheet. The vapor of slaked lime may be substituted for steam of alkaline water or alternated with it. When these methods of treatment are insufficient to relieve an increasing dyspnea the indication for intubation or tracheotomy must be considered and promptly adopted. The former operation should be chosen, if possible, because it is quick, painless, and efficient. Antitoxin and intubation will save four out of every five cases in the later stages of asphyxia. Convalescence will be favored by full doses of tinct. ferri chlor., well diluted with glycerin and water.

Chronic Atrophic Laryngitis.—Laryngitis sicca, or chronic atrophic laryngitis, usually attends similar manifestations in the upper air passages. Pilocarpin in 1/10-gr. dosage, three times a day, by aiding general glandular activity, may prove of service. Phosphorus (gr. 1/100) in oil, in capsule, or iodid of potassium, are also indicated. When a laryngeal spray is used the patient should hold the tongue with a piece of gauze between the thumb and index finger. The operator then holds the nose, and deep inhalations are taken. The use of a menthol (0.30 gm.), albolene (30.00 c.c.), Dobell's solution as a down spray can be used to the larynx and proves grateful. Of the local applications 5 to 10 per cent oleum picis or argyrol (50 per cent) or ichthyol (20 per cent) are most efficient. Numerous remedies have been advocated, such as inhalations of benzoinated steam, applications of (2 to 5 per cent) thymol in petroleum oil, camphor (5 per cent) in oil, zinc sulphate (1 to 3 per cent). When the trachea is involved intratracheal injections of menthol, guaiacol, and camphor in olive oil will prove beneficial. Good results cannot be obtained, however, without careful treatment of the upper passages at the same time (see Atrophic Rhinitis). Hypodermics of the green citrate of iron have a useful tonic action.

TUBERCULOSIS OF THE LARYNX

Treatment may be classified as prophylactic, hygienic, climatic, specific, symptomatic, local, surgical, and general. There is a popular belief among the laity that so-called catarrhal affections of the nose, throat, and larynx lead to tuberculosis. It is exceedingly difficult to prove the scientific truth or falsity of this assertion. It must be remembered that the laryngeal tuberculosis is practically always an infection secondary to a pulmonary focus.

The prophylactic treatment of this affection is grouped around our conception of that which will best promote immunity. This involves the problem of the fight against infection and the predisposing factors of heredity.

Prophylactic treatment is particularly the field of the family physician, who becomes necessarily a student of a predisposed individual. If the treatment by fresh air, sunlight, forced feeding, and carefully directed exercise has any curative value during the progress of the disease it must in all certainty have untold value along prophylactic lines.

Children who bear the earmarks of tuberculous heredity, who are exposed at home to infection, and who show well-defined signs of latent danger, should receive the timely advice of the family physician as to occupation, habits of life, diet, and general hygiene. It is recognized that the difficulties of good results are extremely numerous among a class of individuals who take unkindly to discipline; but the problems of prevention cannot be solved without early diagnosis by the medical profession and painstaking care of the subjects under exposure.

The hygienic treatment requires proper clothing, sufficient but not too frequent bathing, digestible and nourishing food, out-of-door life with properly ventilated or open-air sleeping apartments. Coal gas, air laden with organic matter as found in our cheap theaters and public halls, the use of tobacco, ice, and alcoholic drinks are irritating to these lesions. The use of the voice in laryngeal phthisis should be prohibited or moderated. This rule of rest or exercise is as important in laryngeal involvement as in the pulmonary varieties.

The influence of climate is well recognized by the profession and laity. In laryngeal infections that complicate a far-advanced pulmonary involvement, advice to remain at home is much more valuable than a recommendation to seek some indefinite place in the western country. Far-advanced tuberculosis should be fought at home, and no patient should be allowed to seek new environment without sufficient financial resources. Usually a warm moist climate improves the inflammatory and catarrhal condition of the larynx, but as the pulmonary lesions do better in a dry, warm climate with altitude it is advisable to study the condition and, when possible, recommend sanatorium treatment in New Mexico, Arizona, or selected parts of Southern California. It is essential that medical supervision should be obtained for each patient, and the family physician or specialist who firmly believes that climatic treatment offers for his special patient something of genuine benefit should direct him with great care to some competent practitioner or chief of a sanatorium in the region especially indicated. When the local lesion is in the early stage, these patients do well at a considerable altitude (6,000 feet).

Many cases under careful supervision improve satisfactorily in the lower altitudes—below three thousand feet—at which place they must receive the full benefit of all therapy other than climate. Discomfort and distress are increased during the wind and dust storms of some of our favorite Colorado and New Mexico resorts.

Some remarkable arrests and cures have been obtained among laryngeal and pulmonary patients sent to an altitude such as Silver City, New

Mexico, about six thousand feet. In this region the altitude is played against the latitude, resulting in cool or cold nights with warm or hot days.

Patients with tuberculosis of the upper air tract without much pulmonary involvement will improve and obtain great comfort from an ocean voyage to the Riviera or Egypt.

The numerous specifics that have been brought forward from time to time for the cure of the various forms of tuberculosis have failed to establish the claims made for them. Skepticism and therapeutic nihilism are unjustifiable, however, as usefulness may be found for many of the remedies, in selected cases. Unfavorable results in therapeutic procedure in tuberculosis of the upper tract are largely due to the fact that many of these manifestations, and the laryngeal forms in particular, are almost invariably attended by pulmonary infection of more or less virulence. In a large experience during twenty-five years the writer has not seen a case of primary laryngeal tuberculosis in private practice.

Among the modern so-called specifics may be mentioned tuberculin in various forms, with or without vaccines, sera and antitoxins, iodin, chlorid of gold and sodium, creosote, formalin, oil of cloves, and nuclein. In addition may be mentioned the X-ray and radium.

While brilliant results have been obtained when these sera or drugs were used, the test of time has proved their failure. It is very important to differentiate between tuberculous, chronic, and syphilitic laryngitis before a definite plan of treatment is adopted.

Direct sunlight to the larynx stands out preëminently as the most useful remedy discovered up to the present time. A special apparatus modified by Forster of Colorado Springs consisting of a laryngeal mirror and an adjustable looking-glass with a bracket that may be attached to the back of a chair allows the rays of the sun or those of a suitable 1,500-watt lamp to be reflected directly into the larynx. The patient treats himself daily beginning with a one-minute exposure and increasing with the tolerance of the larynx. Tuberculin may be used hypodermically. The selection of the dose, a study of the individual, a most watchful care of the patient, and careful attention to the rules of administration are essential to successful treatment. Pottenger shows that we have manifest evidence of its value in tuberculous laryngitis, where in small infiltration and even ulceration great improvement and cure may be looked for in a large percentage of cases. Trudeau advances important rules for consideration. He advises a minute dose of 1/10,000 or 1/20,000 mg. of the solid substance of Koch's bacillus emulsion or 1/1,000 mg. of old tuberculin, increased very gradually, and at intervals that will produce as little disturbance as possible. At the slightest evidence of intolerance, such as irritation at the site of injection or slight temperature reaction, the interval should be lengthened and the dose diminished. No injection should be given for some days following a reaction. It is necessary to have the patient under observation for at least six months if this treatment is commenced.

In the writer's experience many cases have been favorably influenced by the use of Deny's filtrate (B. F.), beginning with 1/50,000 or 1/100,000 mg. and increasing with extreme care at weekly or biweekly intervals to avoid every possibility of a reaction. Failure comes from "beginning treatment with too large amounts; raising the dose too rapidly or at too short intervals; repeating the dose before all effects of a reaction have passed; increasing a dose after a reaction; neglecting malaise, headache, anorexia, increased temperature, cough, and expectoration as indications of a limit of tolerance." When a reaction occurs or when anemia is associated with the tuberculous lesions valuable assistance during the interval may be obtained by the hypodermic use of the green citrate or iron (gm. 0.05) and sodium arsenate (gm. 0.001). This method of medication saves a delicate digestive apparatus for other usefulness, and affords a stimulating and reconstructive treatment of great and as yet unrecognized value.

In cases mixed with syphilitic infection mercury and iodin will furnish the best results. The biniodid of mercury (gr. i), potassium iodid (ʒss), cinnamon water, and distilled water (℥ii) each in teaspoonful doses three times a day, will produce a startling effect in properly selected cases. Its administration in lesions of strictly tuberculous type is often detrimental and contra-indicated. The use of phosphorus (gr. 1/100) in 10 to 15 minims of olive oil in capsule is beneficial in the more chronic cases. It should be given after a full meal and promptly discontinued if irritation of the stomach, urticarious eruption, or aphrodisiac effects follow its use. Symptomatic remedies are necessary to allay cough, temperature, pain, diarrhea, and mental distress from time to time. Heroin (gr. 1/12) or codein (gr. ⅛ to ⅙) are efficient drugs to relieve the irritation of the larynx. They should be administered with caution and not until decidedly indicated. Aspirin, guaiacol, and quinin will control temperature when necessary. If alcohol is demanded the malt beverages and preparations other than spirituous are preferable. Rectified spirits in milk is better than whisky, brandy, or rum. While in diarrhea and pain, opium or morphin may be necessary, their use should be postponed as long as possible. Sleep may be secured by veronal or trional if absolutely indicated.

The prescribing of depressing drugs should be withheld from patients who offer a hope of cure, as the resistance may be undermined by the prostration, interference with elimination and normal digestion that attend their constant use.

The local treatment consists of the inhalation of gases or medicated air in sprays or powders and the application of drugs to the affected region.

Among the many ingenious methods of local therapy that have been advocated, a few useful palliatives may be mentioned. Reference may be made to some special textbook if the complete list is desired.

Before treatment is administered, a spray of Dobell's solution should be used to remove secretion from the larynx. The application of formalin in gradually increasing strength is preëminently the most beneficial remedy. It should be prepared daily from a 40 per cent solution and the initial strength should be 3 per cent in glycerin. Until the tolerance of the larynx is obtained the pigment may be used in ½ per cent strength, increasing to one and gradually to 10 per cent. Two per cent cocain may be used if much pain is encountered. The preparation of Lake combines formalin (7 per cent), lactic acid (50 per cent), glycerin (20 per cent), water (to 100 per cent). The application of lactic acid (20 per cent) is a favorite with many laryngologists. Iodoform insufflation is recommended highly by the older laryngologists. Orthoform and anesthesin are used for the relief of pain.

Freudenthal highly recommends fulguration for the cure of ulcerated areas. Argyrol (20 per cent) is an acceptable substitute when irritation follows the use of formalin, lactic, nitric, or hydrochloric acid. Intralaryngeal injections of guaiacol (2½ per cent), menthol and camphor (āā gr. ii) in olive oil are often beneficial in the relief of pain and cough. Deep injections of alcohol into the region of the nerves may be used to allay pain. The use of the X-ray is followed by dangerous reactions.

When these measures are not sufficient to modify or allay the ravages of disease, surgical intervention may be required. The lesions affecting the epiglottis may be removed by epiglottectomy as practiced by Lochard of Denver.

Curettage has been employed to remove the tuberculous deposits. It is possible, however, to remove only a small portion of pathologic tissue by this method. It is advisable only in selected cases where other methods have failed.

When deglutition becomes very painful a spray of cocain (10 per cent) before meals is justifiable. The Wolfenden position may be assumed with additional comfort in swallowing. The head is hung over the bed and the liquid drawn through a tube from the glass upon the floor. In this manner the patient drinks like a horse.

Laryngectomy, thyrotomy, or tracheotomy are rarely indicated in this disease. In the late ulcerative stage it may be necessary to administer an occasional small dose of morphin hypodermically. Constitutional treatment may be of advantage in the earlier stages, in the form of cod-liver oil, malt, hypophosphites, or creosote.

SYPHILIS OF THE UPPER RESPIRATORY TRACT

Modern diagnostic methods of recent years, which include the recognition of the Spirochæta pallida and the deductions from the Wassermann reaction, have materially aided early specific therapy in luetic disease. The treatment consists of both local and general measures.

The general treatment may be commenced with the definitely recognized chancre. It consists essentially in the administration of well selected preparations of mercurials and iodin and the use of arsphenamin. The choice of preparation may be determined by the character and stage of the lesion. A definite plan of procedure should be laid out for each patient. For the primary and secondary forms mercurials predominate. We must decide upon a choice and dosage of hypodermic medication by salvarsan, intramuscular or intravenous, the hypodermic method of adminstration of mercury bicyanid, bichlorid, biniodid, salicylate, or calomel; the method by inunction, which consists of blue ointment, mercury vasogen, mercurettes, or the administration by mouth of blue mass, calomel, mercury protoiodid, bichlorid, etc. It is important to adopt one of these definite plans and adhere to it until the indications for a change of method or preparation are demonstrated.

In the later stages of the secondary form the mixed treatment is more efficient. This consists of a prescription containing the bichlorid or biniodid of mercury with potassium iodid.

℞	Hydrarg. biniod.	gr. i
	Kalii iod.	ʒss
	Aq. cinnamomi	
	Aq. dest.	āā ℥ii
Sig.	ʒi tid. p. c., c. a.	

When the disease is very active or the individuals under observation are particularly irresponsible or careless with the directions imposed, it is necessary to use arsphenamin or other hypodermic medication at once.

Before the use of salvarsan is undertaken a careful study of the patient with special reference to the condition of the heart, lungs, kidneys, and acoustic nerve should be made. It may be wise to explain possible dangers in its use and, if the intramuscular method is chosen, the fact that the injection is often quite painful should be mentioned. The amount of pain will depend upon the care with which the salvarsan is prepared and the site and method chosen for the injection. The average dose for intramuscular injection is 0.6 gm. (9 gr.). When the intravenous method is selected the average dose for women is 0.3 gm. (4½ gr.) and for men 0.4 gm. (6 gr.). It is often necessary to repeat this dosage a second or third time, at intervals of one to four weeks. In feeble patients caution should be observed, and a small dose of 0.2 to 0.3 gm. (3 to 4½ gr.) administered. This may be repeated in a couple of days.

For children 0.2 gm. and for infants 0.02 to 0.05 gm. are advised.

It is exceedingly important that the chemist or pharmacist should prepare arsphenamin for use with the greatest care and detail. Instructions prepared by Elsner and others are readily obtained.

The marvelous and astonishing results in lesions of the upper respiratory tract are second only to those from the use of diphtheria antitoxin.

The local auxiliary treatment for primary infection should consist of mild, cleansing topical applications with a view to removing adherent secretion and modifying the character of the local disturbance. Sprays of peroxid of hydrogen, Dobell's solution, or 1:4,000 bichlorid are useful.

In the ulcerative forms, permanganate of potassium, 2 gr. to the ounce, may be chosen with advantage. Nitrate of silver (10 to 30 gr. to the ounce) or the insufflation of iodoform, aristol, or calomel may be used with advantage. In sluggish ulceration lactic acid (20 per cent) or trichloracetic acid may be used to sear over the diseased area. The general hygiene, and especially the toilet of the mouth, should be strictly enforced. Frequent simple or hot saline baths aid materially the action of mercury and the iodids.

If the hypodermic use of mercury is desired a painless preparation of 1/20 to 1/40 gr. of cyanid of mercury may be obtained. Hypodermic iodin (5 to 10 to 20 per cent) in almond oil (iodolaine) may be used also when patients fail to take these remedies well by mouth.

In the treatment of congenital syphilis no preparation is more satisfactory than mercury with chalk. In infants the body band may be smeared with mercurial ointment daily.

The sequences of syphilis demand special treatment to suit the individual case. Impending stenosis must receive daily dilatation by properly selected methods and instruments. Laryngeal stenosis may require intubation, tracheotomy, or the passage of Schroetter's tubes. A few experts in the rhinolaryngological field obtain good results in the surgical relief of old cicatricial deformities. In cartilaginous and bony necrosis the sequestra should be removed at the earliest moment of sufficient separation. The relief of "saddle-nose" may be obtained by the use of paraffin or suitable operative procedure. Gummata or ulceration in the trachea may be examined by the Jackson tubes and appropriate local treatment applied.

CHAPTER III

DISEASES OF THE BRONCHI

Robert D. Rudolf

BRONCHITIS

Acute Bronchitis

Acute catarrh of the respiratory tract is usually a widespread affection, involving more or less of the mucous membrane which extends from the upper air passages to the point where the bronchioles terminate in the air vesicles of the lung. The catarrh may not stop here even, but may involve the air vesicles themselves, producing the common complication of capillary bronchitis—catarrhal pneumonia.

Acute bronchitis is not a specific affection, but may be due to divers causes, and before attempting to take charge of a case it is necessary to endeavor to find out the exact etiological nature of it. Is it a primary infection of the respiratory mucosa; is it a local manifestation of some general infection such as measles or typhoid fever; or is the catarrh secondary to a condition, such as mitral stenosis, congenital heart trouble, or some toxemia, such as gout, uremia, etc.?

General Therapy.—The amount of care necessary in a given case of catarrh of the respiratory tract depends upon two things: (*a*) the severity and extent of the inflammation; (*b*) the general condition of the patient. Thus, as regards the first factor, the treatment of a cold in the head and a laryngitis is a very much less urgent thing than when the same catarrh reaches down to the bronchioles. Again, as regards the second factor, what would in a healthy adult be a very trifling attack of bronchitis, requiring very simple treatment, may, in the case of a frail and aged person, be attended with the greatest risk to life, and must be combated in every possible way. Many writers classify their cases of bronchitis as regards the therapy into: (1) catarrh of the trachea and larger tubes; (2) catarrh of the smaller bronchi and bronchioles. The division is artificial, as the classes merge so gradually into one another; but, although it is not done in this chapter, the practitioner must use his discretion as to how much therapy a case requires.

Environment.—People with a tendency to acute bronchitis should be carefully guarded against exposure to chills and to rapid changes of temperature, such as the leaving of a heated room for the colder air outside. Often it seems also that hot stuffy air itself is the cause of an attack, even if not followed by exposure to cold, and many people have learned by bitter experience to avoid crowded and heated rooms on this account.

It was noticed early in the late War that the Canadian troops remained well while under canvas during very bad winter weather on Salisbury Plain, but very many suffered from bronchial catarrh as soon as they were housed in huts.

Patients, however, should be cautiously hardened against the evil effects of chill by free exercise in the open air and by the careful use of cold baths and sponging followed by a rapid and vigorous rubbing with a coarse towel. Salt baths are here specially useful, the salt seeming to have a stimulating effect upon the skin, thus promoting a good reaction. To make a salt bath, rock salt should be added to the water to make a 1 per cent solution. The strength may be increased to 5 per cent if the reaction from the weaker bath be not sufficient. If a cold bath or sponging is doing good, it should be followed by such a reaction as to produce a sensation of glowing; if this does not occur, but instead the patient remains shivery, with "goose skin" and bluish extremities, then the cold water should not be used, being replaced by a warmer solution. A short immersion in really cold water will often produce a better reaction than with a longer one in less chilly water. As a rule the bath should be between 65° and 75° F.

It often becomes necessary for people with a tendency to bronchitis to avoid the winter months in a cold climate altogether. Where this is possible, they should select a place having a warm and equable climate.

The spring is usually the most trying time for such people, and one frequently sees the mistake made of their returning to their northern homes too soon, after a winter spent in the south, with a resulting attack of acute bronchitis. No sufferer from bronchitis who has gone south for the winter should return until all probable danger of cold weather has passed. It is a good plan for them to come gradually north, stopping occasionally for a few days at intermediate places, rather than to do the long journey in one stretch.

By thus avoiding the winter for one or two seasons, predisposed individuals may go through the trying cold season without an attack.

Where, for financial or other reasons, it is out of the question for the individual to go south, then it may be necessary for him to remain indoors during the cold months. This point will be further discussed under the heading of Chronic Bronchitis.

Nothing predisposes so much to an attack of acute bronchitis as a previously existing chronic inflammation of the mucous membrane, and hence a patient with this chronic condition should be specially looked after and guarded against any of the causes of an acute attack. When he goes into the cold air he should be cautioned to breathe through the nose, and the wearing of a respirator is often advisable, especially in the aged. Where the upper air passages are the seat of adenoids, enlarged tonsils, or nasal polypi, these abnormalities should be attended to and if necessary removed, as they cause mouth-breathing and, further, are apt to be the local cause of a catarrh, which may then become more widely spread. The nasal passages are the natural warmers of the inspired air (see Chronic Bronchitis).

An individual actually suffering from acute bronchial catarrh should be confined to bed, and he should remain there as long as there is any fever. In such an environment he will get over the attack more speedily than he will otherwise do, even if he stays in one room. Even where the catarrh is limited to the upper air passages, recovery will be more speedy if the patient takes to his bed for a day or two.

The air of the room should be kept fresh by free ventilation, but should not be cold, and the patient should be kept out of draughts. Many modern clinicians believe that the temperature of the sick room does not matter much so long as the air be pure, but, in the writer's opinion, bronchitic cases should not be exposed to such cold as may with impunity and even benefit be allowed in cases of lobar pneumonia and pulmonary tuberculosis. Cold air acts as an irritant to the inflamed bronchial mucous membrane, apart from any danger of chilling of the surface of the body, and a bronchitic patient soon finds how a breath of cold air makes him cough.

An atmosphere at a temperature of about 65° F. is probably the best, and care should be taken that the room does not become much colder than this. The greatest risk of this is in the early hours of the morning, when the furnace is apt to become low.

A Turkish bath will sometimes cut short an acute bronchial catarrh if taken at the very beginning of the attack. Short of a Turkish bath, a thorough sweating, produced by the use of a hot-air bath, may act in the same beneficial way. Folding cabinets for this purpose are obtainable and are often very efficacious. The patient is undressed and wrapped in a blanket and allowed to stay in the hot-air bath for from ten to twenty minutes. While there he should have a cold wet towel round the head and should drink freely of lemonade or some alcoholic drink as directed. The Imperial Drink, made by dissolving a teaspoonful of acid tartrate of potash (purified cream of tartar) in a pint of boiling water and then adding a slice of lemon peel and sugar to taste, and taken cold, is useful here as a diaphoretic and diuretic, and is very refreshing to the fevered patient.

The Turkish bath, or hot-air bath, should only be used where the patient can go at once to bed, and if this be impossible it is best not to use either at all. Many an individual gets more harm than good from taking a Turkish bath and then going out in the cold soon after it.

A moist, warm atmosphere has a soothing effect upon the inflamed mucous surfaces, and, in severe cases, it is well to surround the patient's bed with a tent, into which steam is introduced.

The steam may be had from a "bronchitis kettle" made for the purpose, but one may be easily improvised by attaching a cone of brown paper or thin cardboard to the spout of a kettle. The steam should not be directed toward the patient's face, but rather in the direction of the foot of the bed. This is a practical point which is often neglected, with the result that the respiratory tract is irritated by a too hot vapor.

The patient should be kept very quiet, the act of speaking often being sufficient to produce a fit of coughing. With this end in view it is often wise to exclude friends.

In the dry, irritable stage of the disease, especially when the larynx is involved in the catarrh, the act of coughing tends to further irritate the mucous membrane, and thus a vicious circle is established. To tell the patient not to cough is often a counsel of perfection, and yet, by getting him so to endeavor, many unnecessary spells of coughing may be avoided, and thus the inflamed parts are at least partially rested.

In severe cases the calls of nature must be attended to in bed, and in all cases the patient should be specially protected from chills when attending to these needs.

Every sufferer from bronchitis has *some* dyspnea, and this may be so marked that he may require to be propped up in bed. As he improves this propping up will be less needed, and the fact that to-day he can do with a pillow less than yesterday is one of the most certain signs of improvement.

In severe cases the patient should not be left in one position too long, and the nurse must be instructed to shift him from side to side at frequent intervals in order to lessen the chances of circulatory and bronchial *stasis* in any one part of the chest.

William Ewart emphasized the value of mechanically aiding expiration in cases of acute bronchitis complicating emphysema. The expiration, which should normally be a passive act, becomes, as a result of the loss of elasticity of the lungs, an active one, and hence tends to tire the patient. The trained nurse, or the physician, should place the hands over the axillary bases of the lungs and exert carefully timed pressure here, synchronously with expiration. The effect is to tend to eject some of the secretion further up the respiratory tract, whence it may be more easily coughed up. The method is specially valuable and necessary in the aged and feeble. It should be repeated for a few minutes several times in the twenty-four hours.

Diet.—Patients suffering from acute bronchitis have fever, and on general principles their diet should be light and digestible, as here, as in all cases of fever, the digestive powers are more or less limited. Milk should be the staple food, and, in addition, milk puddings, gruel, jellies, soups, and other light things may be added.

Warm mucilaginous or slightly alkaline drinks are comforting to the patient, and in the early and dry stage of the catarrh act as expectorants, diminishing the tenacity of the sputum and in this way aiding the cough. Barley water, linseed tea, thin gruel, and oatmeal water are all useful here, and soda or potash water with milk is an excellent drink.

The addition of some whisky, cognac, or rum to such drinks at bedtime in many cases, especially with aged patients, secures a better night's rest.

Although a largely fluid diet is generally advisable in acute bronchitis, it may occasionally be wise to allow adult patients, who happen to dislike fluids, to have more solid articles, such as lightly boiled eggs, puddings, with bread and butter.

In the slighter cases of acute bronchial catarrh a hot bath, a hot bed, a hot drink of gruel and whisky, with 10 gr. of Dover's powder at bedtime, may be sufficient to bring him back to the normal by next day.

If there be any constipation it is well, at the beginning of the attack, to give a mercurial purgative, such as calomel or blue pill, followed in either case by some saline next morning.

Specific Treatment.—Bronchitis is not due to any one infection as a rule, and it is usually beyond our powers to attack it by any specific therapy. Within recent years various vaccines have been used, but these have chiefly been employed in the more chronic and persistent forms of the disease (see Chronic Bronchitis).

Camphor.—Many people think, and probably with some reason, that they can cut short or lessen a respiratory catarrh by the early and free use of camphor internally. One gr. (0.066 gm.) may be given every two hours or so, either by itself in pill or capsule form, or it may be combined with other ingredients. A useful prescription is one suggested by Dr. Arthur Newlin, as follows:

℞	Codeinæ sulphatis	gr. 1/16	0.004 gm.
	Camphoræ monobromatæ	gr. 1/2	0.030 gm.
	Aspirin	gr. v	0.300 gm.

Misce et fiat capsula.

Sig. One capsule to be taken every three hours.

Quinin.—Quinin is much used by the laity for checking a catarrhal fever, and seems to have some good effect. A useful prescription in feverish cold is as follows:

℞	Quininæ sulphatis	gr. i	0.06 gm.
	Phenacetini	gr. ii	0.15 gm.
	Sodii salicylatis	gr. iii	0.20 gm.

Misce et fiat capsula.

Sig. Take one capsule every four hours.

It is often a good plan to give two of these capsules for the first dose and then follow with one every four hours.

Symptomatic Therapy.—The natural tendency of an acute respiratory catarrh is to disappear, and, in most cases, as already said, if the patient be kept warm and at rest, the disease will more or less rapidly go through its phases of congestion, secretion and final drying up of this, and will leave the mucous membrane none the worse for the invasion. In coryza, where inflammation of a mucous membrane may be watched and directly studied, the first stage is seen to be one of engorgement, the membrane being swollen and reddened, so that more or less complete obstruction of the nasal passages occurs. Next follows the stage when there is free secretion of a watery exudate, which is accompanied by great relief of the engorgement and consequent ease to the sufferer. In the third stage the secretion become scantier and mucopurulent, and then gradually dries up and disappears. In catarrh occurring further down in the respiratory tract very much the same stages are gone through. As a result, the patient first has a dry and irritable cough, with some dyspnea due to obstruction from the swelling of the mucous membrane. There is usually also substernal discomfort and even pain from the same cause. Next, as the secretion becomes established, the cough grows freer and productive of a thin and often frothy expectoration. Finally this secretion becomes thicker and scantier and then disappears.

Much may be done by appropriate treatment to lessen the patient's discomfort as he passes through these stages, and, by a proper medicinal and other therapy, we are often able to hasten his return to the normal, and even to bring this about when otherwise it might not occur.

At the onset of every acute respiratory catarrh, as already mentioned, it is a wise plan to give a purgative. A useful one is the following:

℞ Pilulæ hydrargyri
Pilulæ colocynthidis et hyoscyami āā gr. ii to v (0.15-0.35 gm.).
Misce, fiat pilula.

Sig. One at bedtime, followed by a Seidlitz powder in the morning.

Or the following:

℞	Pilulæ hydrargyri		
	Extracti colocynthidis compositi		
	Pilulæ rhei compositæ	āā gr. 1½	0.10 gm.
	Extracti hyoscyami	gr. ½	0.03 gm.

Misce, fiat pilula.

Sig. One at bedtime, followed by a saline next morning.

Or the patient may be given ½ to 2 gr. (0.03 to 0.12 gm.) of calomel at bedtime, with a saline next morning.

Local Treatment—*Linseed Poultices.*—In the early stage of acute bronchitis the application of *linseed poultices* to the chest gives relief. The poultices must be light, and as hot as can be borne, and, if properly made, are a great comfort to the patient. But a badly made, perhaps lumpy, heavy, and half-cold poultice is worse than useless, and if we cannot be sure of having them well made we had better do without them.

Poultices should be renewed every two hours. It is often a good plan to put one on for two hours, then replace it by a cotton wool jacket for two hours, then a poultice again for two hours, and so on.

Mustard Poultices.—A mustard poultice over the upper sternal region is often useful. It may be left on for ten to twenty minutes, and upon removing it the skin will be found to be reddened, and should then be smeared with vaselin. On no account should the poultice be left on indefinitely, or, as one sometimes hears, for "as long as the patient can stand it," for after some minutes the oil of mustard acts as an anesthetic and, without much discomfort to the patient, a severe burn may be inflicted. Slight burns from this cause are commonly seen, but very severe ones may occur, as in a case seen by the writer, where an infant was so badly burned that it died, and at the *postmortem* it was found that the ribs and sternum were included in a deep slough.

Mustard Leaves.—"Mustard leaves" are used in the same way, left on for the same time. They are less severe than the poultice.

Turpentine.—Turpentine is a favorite external application in acute bronchitis. It may be sprinkled upon a cloth, which is then applied for ten to twenty minutes, or it can be used in the form of a *turpentine stupe,* which is a hot fomentation with a teaspoonful of turpentine sprinkled over it. The stupes should be renewed every four hours, and after the first one or two the turpentine is best omitted.

Stimulating Liniments.—In the slighter cases of acute catarrh, and in most cases later on, *stimulating liniments* are valuable. They are usually rubbed over the whole chest night and morning and, by keeping the skin reddened, seem to relieve the deep congestion.

Turpentine is the drug most commonly used for this purpose, either in the form of the *linimentum terebinthinæ,* or, if this be not strong enough to redden the skin, then as the *linimentum terebinthinæ aceticum* (*B. P.*). The latter is an imitation of the celebrated St. John Long's Liniment. A useful addition to the ordinary turpentine liniment is the one suggested by Kingston Fowler, which consists in the adding of a drachm of the tincture of iodin to each ounce of the liniment. In severe cases, where the dyspnea is marked, dry, or even wet, cupping over the roots of the lungs in front and behind is a useful measure.

Leeches.—The application of six or eight *leeches* over the roots of the lungs is often valuable in such cases. Where leeches are used it must, of course, be seen that the bites have stopped bleeding before the patient is left, as neglected leech bites may bleed very severely. The bleeding is easily stopped, as a rule, by the application of powdered alum, over which pressure is applied by means of tightly rolled absorbent cotton kept on with strapping. *Dry* or *wet capping* of the chest is often useful. It is a remedy much used in France.

Cold.—Some practitioners prefer to apply cold rather than heat to the chest in acute bronchitis. An ice-bag may be used, and it is necessary to see that this is only partially filled with finely broken ice, and that all the air is excluded before the bag is closed. If these small details be not attended to, the bag does not lie comfortably on the chest, or indeed on any part of the body. Or cold may be applied in the form of a cold compress. Such compresses should be changed rather frequently, say every fifteen or thirty minutes, but even if left on for several hours seem to give relief; as long, indeed, as any moisture remains in them.

The Priessnitz compress, so highly recommended by Ortner of Vienna, consists of a cloth wrung out of water at room temperature, with a hot cloth bound over it. As soon as the inside compress is dry it should be renewed, and after it is finally removed the chest should be thoroughly rubbed, sometimes after it has been first sponged with cold water. As Ortner says, the action of this compress is not exactly understood, but it appears to be somewhat as follows:

The cold application at first causes an active contraction of the cutaneous vessels. As soon as the cold bandage is warmed by the application of the hot one over it, this contraction gives place to active dilatation combined with a sustained tone of the vessels, which persists for some time and causes an active hyperemia of the skin. By the initial application of cold the nervous centers are reflexly stimulated, the breathing is momentarily deepened, and the nervous tone of the vessels is increased. By the alternate action of the heat and cold the innervation of the internal organs is stimulated reflexly and the secretion of the diseased bronchial mucous membrane and the circulation in the lungs are favorably affected.

In some cases, owing to the low tone of the patient, the cold compress remains cold even after it is covered by the hot bandage, and the patient experiences an unpleasant feeling of chilliness until it is removed. In this case it is well to use, instead of the outer flannel, a piece of oiled silk or rubber tissue. This will probably prevent the feeling of chilliness.

Vapors.—There are many means by which *applications* are directly made *to the inflamed mucous surface.* The most common one perhaps is the inhalation of vapor from hot water, either alone or impregnated with some medicinal agent. As already stated, when it is thought necessary to use a steam kettle, the spout, or cone connected with the spout, should not be directed toward the patient's face, but rather toward his feet, when the patient is in bed with a tent over it. *Compound tincture of benzoin*—Friar's Balsam—is commonly added to the water in the kettle, a teaspoonful or so being put in, and as a result the vapor becomes loaded with the volatile ingredients of the preparation. In slighter cases vapor may be inhaled from a jug over which a folded towel is so placed as to direct the vapor toward the face. The water in the jug should not be at boiling point, but at a temperature of about 150° F. This temperature may be roughly attained by mixing equal parts of boiling and of tap water. On the surface of the water may be thrown Friar's Balsam or any other medicament which it is desired to use. Such moist inhalations have a very soothing effect, and may be repeated with advantage every hour or so, each period of inhalation lasting for about five minutes. A pleasant preparation to be used in this manner is one recommended by P. Claisse, as follows:

℞	Menthol	gr. i	0.060 gm.
	Tincturæ benzoini	min. x	0.600 c.c.
	Olei eucalypti	min. v	0.350 c.c.
	Alcoholis (90 p. c.)	ad dr. i	4.000 c.c.

Misce, fiat mistura.

Sig. This amount to be placed in a little hot water and inhaled for five minutes.

Sprays.—Sprays are often valuable in acute respiratory catarrh, especially when inflammation involves the trachea and mucous membrane higher up than the bronchi. Seiler's solution may be used here, or a spray containing the following: sodium bicarbonate, 10 gr. (0.600 gm.); glycerin of carbolic acid, 1 dram (4.0 c.c.), in an ounce (30.0 c.c.) of rose water. There are a great variety of such sprays used by the profession, the chief points of similarity between them being that they are alkaline and contain some aromatic antiseptic. Such sprays should be used warm and should be frequently repeated.

In the later stages of acute catarrh the inhalation of *eucalyptus* appears to lessen the secretion and give comfort to the patient. He may inhale the pure oil from a handkerchief or from a Burney Yeo zinc inhaler. A valuable mixture to be thus used is the following:

℞	Menthol	1 part
	Eucalyptol	2 parts
	Benzoinol	3 parts

Misce et fiat mistura.

Sig. Inhale frequently.

An even more soothing inhalant contains equal parts of menthol, eucalyptol, terebene and chloroform.

Turpentine may be inhaled in the same way, and is often useful in the later stages of a catarrh. It is too irritating for some patients, and may then be advantageously combined with equal parts of spirit chloroform.

Internal Medication—*Laxatives.*—As already stated it is well to begin the treatment of a case of acute bronchitis with a laxative.

Many drugs are used with the objects of lessening the fever and modifying the bronchial secretion.

As regards the latter indication, the bodies chiefly used in the early stages of the disease belong to the class of so-called soothing or nauseating expectorants, of which class ipecacuanha, antimony, and various alkalis are the chief members.

Expectorants.—The whole subject of the use of expectorants is at present in a state of chaos. From a pharmacological point of view many of the drugs which clinicians have found to be of most value are without expectorant effect, while, on the other hand, others which are most effective according to laboratory experimental evidence do not find favor with the practitioner. Probably a reason why these differences of opinion exist is that the two classes of observers are dealing with different tissues, the clinician using diseased subjects for his work, while the pharmacologist does most of his experiments upon normal animals or people. A diseased mucous membrane may react quite differently to a normal one. Moreover, the reports of different pharmacologists upon the expectorant action of the same drugs differ widely in many cases; so much depends upon the way in which the experiments are done, the dose used, the kind of animal employed, the anesthetic given, etc.

It must for the present be admitted that the employment of drugs in the treatment of bronchial affections is more of an art than a science. No doubt the tendency has been in the past to use expectorant mixtures with too free a hand, but they have their place, and, when skilfully employed, undoubtedly decrease the discomfort of the patient and hasten recovery.

F. Forchheimer expressed the opinion of most thinking clinicians when he said: "Experiment has taught me that the expectorants are unreliable, but I should not like to be without them."

In a mild case of acute bronchitis the following mixture will promote the patient's comfort and hasten the onset of free secretion:

℞

Vini ipecacuanhæ		
Potassii acetatis	āā dr. i	āā 4.00 gm.
Spiritus ætheris nitrosi	dr. ii	8.00 c.c.
Syrupi tolutani	oz. ½	15.00 c.c.
Liquoris ammonii acetatis	ad oz. iii	100.00 c.c.

Misce, fiat mistura.

Sig. A dessertspoonful in water every four hours.

If the cough be very irritating a little opium may be added to each dose—for example, 10 drops of the camphorated tincture of opium (paregoric).

Antimony acts powerfully as a depressing expectorant. It is usually given as the wine of antimony, and quite small doses frequently repeated are useful, and, in fairly strong patients, are free from depressing effects.

℞	Vini antimonii	dr. i to ii	4.00 to 8.00 c.c.
	Liquoris morphinæ hydrochloridi	dr. ½	2.00 c.c.
	Liquoris ammonii acetatis	oz. ii	60.00 c.c.
	Syrupi tolutani	oz. ½	15.00 c.c.
	Aquæ chloroformi	ad oz. vi	ad 200.00 c.c.

Misce, fiat mistura.

Sig. A tablespoonful every four hours.

In sthenic individuals with high fever *aconite* is often used. It is best given in small, frequently repeated doses; thus 1 drop of the tincture (B. P.) is given every fifteen minutes until the skin becomes moist and the pulse slower. Then this may be replaced by a mixture containing ipecacuanha or antimony, such as one of the above. It should be noted that the American tincture of aconite is double the strength of the British. It is well to remember that preparations of aconite quickly deteriorate and even with good preparations the effect is very uncertain. The action of aconite, however, is very slight and the writer found that even much larger doses than pharmacopeial ones failed to affect the pulse rate or blood-pressure.

Apomorphin is used as a depressing expectorant in the early stage:

℞	Apomorphinæ hydrochloridi	gr. ½ to ⅔	0.02-0.04 gm.
	Acidi hydrochlorici diluti	dr. ½	2.00 c.c.
	Glycerini	oz. ½	15.00 c.c.
	Aquæ destillatæ	ad oz. vi	200.00 c.c.

Misce, fiat mistura.

Sig. A tablespoonful every two to four hours.

Pilocarpin is used a good deal in Germany for increasing the flow of bronchial secretion in early cases. It should be given in doses of 1/20 to 1/10 gr. (0.003 to 0.006 gm.) thrice daily in the form of the nitrate by mouth, or hypodermically in rather smaller doses.

The use of *opium* or its preparations in acute bronchitis requires great care and discrimination. When used in the dry and irritable stage of the disease, in small doses, it is often of the greatest value in easing the wearing and useless cough; but it should never be given in cases attended with a profuse secretion, and in suffocative ones. In such patients we dare not lessen the sensitiveness of the mucous membrane and thus reduce the cough upon which the patient's very life may depend.

℞ Extracti opii	gr. ⅛	0.008 gm.
Extracti stramonii	gr. ⅙	0.010 gm.

Misce, fiat pilula.
Sig. One every two hours.

Codein is often a useful drug where an opium preparation is indicated as it disturbs the alimentary functions less than does opium or morphin. It goes well with ipecacuanha wine and spirit of chloroform:

℞ Codeinæ phosphatis	gr. vi	0.400 gm.
Vini ipecacuanhæ	dr. ii	8.000 c.c.
Spiritus chloroformi	dr. ii	8.000 c.c.
Aquæ	ad oz. iii	100.000 c.c.

Misce, fiat mistura.
Sig. A teaspoonful every four hours.

Such a prescription would be appropriate in the early and dry stage of the acute disease.

Heroin hydrochlorid (diacetyl-morphin hydrochlorid) is an artificial alkaloid prepared from morphin, and has been advocated as a substitute for morphin in respiratory conditions. It may be given in doses of 1/24 to 1/16 gr. (0.002 to 0.004 gm.) either by the mouth or hypodermically. [1]

Soporifics.—Sleeplessness is often a marked feature in acute bronchitis and may require to be specially met. *Opium* should never be given in such a case to "obtain a good night's rest." *Veronal* in doses of 5 to 7 gr. (0.300 to 0.400 gm.), *sulphonal* in doses of 15 to 20 gr. (1.00 to 1.30 gm), or *paraldehyd* in doses of ½ to 1 dr. (2.00 to 4.00 c.c.) are preferable. The last-named drug is very valuable as a non-depressing hypnotic. It has a most objectionable taste and odor, but may be fairly easily given as a mixture, flavored with syrup and licorice, or else in capsules. It may also be given by the bowel.

Chloral hydrate is often useful in the sleeplessness of acute bronchitis, but its effects must be closely watched, when it is decided to use it. It is perhaps best to give a small dose, which may be repeated once or twice during the night at the discretion of the attendant. The drug is rapidly excreted and thus will not accumulate in the system. The syrup (B. P.) contains 10 gr. of the drug in each dram, which is an average dose.

Other useful and comparatively harmless hypnotics are *chloralamid,* which produces chloral by its decomposition in the body, and has less effect upon the circulation than has that powerful drug; *trional,* and lastly *bromural.* Chloralamid is given in doses of 15 to 45 gr. in cachet, or dissolved in a weak spirituous or acidulated solution; trional, in 10 to 20-gr. powders or in cachets; and bromural, probably the mildest of all of those mentioned, in doses of 10 to 15 gr.

---

[1] The widespread use of heroin by drug habitués has led to its abandonment in some state institutions as a component of cough mixtures. While its value is not denied, it would seem that the dangers incident to its use are so great that other substances almost equally efficient and less dangerous should be relied upon.—Editor.

If hypnotics require to be used for some time, it is wise to vary them, and as a rule not to give them oftener than every second or third night.

When, in acute bronchitis, the secretion is well established, expectorants are not much required, but many use here, with apparent benefit to their patients, the so-called stimulating expectorants. These give relief and appear to make the expectoration come away with more ease and then dry up rapidly and disappear.

Stimulating Expectorants.—The stimulating expectorants most commonly used are *squills, senega* and *ammonium carbonate.* For example:

℞

Ammonii carbonatis	½ dr.	2.00 gm.
Tincturæ scillæ	3 dr.	12.00 c.c.
Syrupi tolutani	½ oz.	15.00 c.c.
Infusi senegæ	3 oz.	90.00 c.c.
Aquæ chloroformi	ad 6 oz.	200.00 c.c.

Misce, fiat mistura.

Sig. A tablespoonful every four hours.

If the cough be troublesome, a little opium may be added to this mixture, but, as already said, this must be done with great caution, if at all, when the secretion is very free.

Belladonna.—In severe cases of bronchitis, where the dyspnea is marked and there appears to be extensive involvement of the bronchioles, belladonna is valuable, in that it lessens the secretion and at the same time stimulates the respiratory center. The same is true when the bronchitis is complicated by pneumonia, and in such cases it is well to push the belladonna until the pupils dilate and the mouth becomes dry and the skin flushed. In the bronchopneumonia of children this treatment seems to be of special value. When wheezing is a marked feature of a bronchial attack belladonna is useful in relieving this asthmatic tendency.

Sydney Ringer and William Murrell have both very strongly recommended the use of belladonna in the secreting stage of acute bronchitis, and the writer has frequently seen the great value of it here. N. T. Davies, of Sherborne, speaks of its "magic effects" when used in acute bronchitis, and, indeed, in asthma also, as an *inhalation.* He recommends 1 gr. (0.065 gm.) of the extract of belladonna dissolved in half an ounce (16 c.c.) of warm water in a Dr. Siegel's inhaler. The writer has no experience with this method, but it should be valuable, as drugs are very rapidly absorbed from the respiratory tract, and one would get both the local and also the general effects of the atropin.

Lobelia.—The ethereal tincture of *lobelia* is also much used here, in doses of about 10 drops. Its action resembles that of belladonna.

Measures for Relief of Cyanosis—*Cupping.*—When any cyanosis exists the likelihood is that the right heart is overstrained and tending to give way, and remedies must be used for its relief. *Dry cupping* over the sternum and scalpulæ is valuable here. It is indeed a remedy, the good influence of which has been independently discovered by most races the world over. Where the distress is very severe *wet cupping* may be employed. A scarifier is convenient, but by no means necessary, for this, as a few preliminary shallow incisions with a sharp scalpel are all that is required. A Bier cup is specially useful where wet cupping is to be done, as the suction may be regulated in a way that is not possible where the vacuum is obtained by burning spirit. A few leeches over the roots of the lungs act in much the same way. If these milder methods of relieving the cyanosis be not sufficient, then *bleeding* from a vein in the arm, or even from the external jugular vein, may be practiced, with great relief. The removal of 5 to 15 ounces of blood is usually sufficient. In very severe cases the blood may not flow from the opened vein, and it may be then assisted by gentle aspiration with a syringe.

Strychnin.—In these distressing cases hypodermics of strychnin are of great value, and 1/30 gr. (0.002 gm.) may be given every three or four hours with advantage. This dose may be doubled in severe cases.

Inhalation of Oxygen.—The value of this measure is very differently gaged by different observers, but undoubtedly it has some action in lessening cyanosis, and in the writer's opinion should never be neglected in severe cases where this lividity is marked. Ortner says here:

"In cases of dyspnea, cyanosis, marked diminution of the respiratory surface, and coincident weakness of the heart, the above mentioned hydropathic measures may be supplemented by the inhalation of oxygen. If 25 gallons at least are inhaled daily, good results often appear, although we may not be able to explain them theoretically. The breathing and heart action are slowed, and the subjective symptoms are much improved, showing that, owing to the inhalation of oxygen, more oxygen has passed into the blood and tissues."

The method by which oxygen is given is all-important. If the gas be merely administered through a funnel held near the face of the patient little good results. An excellent though wasteful way is to give it through a rubber tube placed in one nostril. If this tube in the nose causes distress it may be put in the mouth. Still better is the Meltzer inhaler. An oxygen chamber is the best of all but naturally can only be employed in large institutions. In severe bronchitis and in bronchopneumonia the arterial blood is undersaturated with oxygen and this undersaturation can be largely removed by an efficient inhalation of the gas.

The idea naturally occurs that in desperate cases oxygen gas might be administered directly into the tissues, and several writers have urged the value of this method. Recently Dr. Sacquépée, professor at the Val de Grâce, read an interesting paper before the Société de Médecine Militaire Française on the use of oxygen in this way in acute respiratory conditions. The treatment is purely symptomatic, but rapid relief of the distressed breathing is seen, the heart beats are slowed and the blood-pressure rises. Rapin mentions a case where a child of fourteen months was apparently *in articulo mortis* from diffuse bronchitis of both lungs. The child was unconscious, cold, cyanosed and the pulse was imperceptible. Various remedies were tried without effect, including the inhalation of oxygen. Then this gas was injected under the skin of the thigh. The immediate effect was to raise the temperature locally, and the lividity of the skin here was replaced by a rosy color. The dyspnea became relieved and the pulse perceptible. The treatment was cautiously continued, first in one thigh and then in the other, and the patient recovered. Experiments on rabbits gave the following results: (1) An animal which has received a prophylactic injection of oxygen under the skin resists asphyxia longer than does a rabbit not so prepared. (2) Existing asphyxia can be relieved by the hypodermic introduction of oxygen, and the pulse simultaneously improves. (3) If asphyxia is pushed until the corneal reflex is abolished, the injection of oxygen permits of recovery, even though the duration of apnea has been prolonged beyond the period fatal in control animals. The method would probably prove of service in any condition characterized by cyanosis and asphyxia. The apparatus is simple: A rubber tube is connected by a stopcock with a gas bag full of oxygen. The other end of the tube is lightly plugged with sterile cotton wool, and then connected with a large-bore hypodermic needle. In France where the method has been much used lately Martinet's oxygenator is usually employed.

Emetics.—Occasionally, when the bronchial secretion is excessive and the power of coughing it up is insufficient, an emetic is valuable and even life-saving. Emetics are chiefly given to children; they should be used with caution where the pulse is weak. Any sign of pulmonary collapse, however, is an urgent indication for such therapy in strong patients. For an adult, 20 gr. (1.35 gm.) of powdered ipecacuanha or half an ounce of the wine will usually be sufficient. Mustard and water is a useful and relatively non-depressing emetic, and should be given in the strength of a tablespoonful of mustard dissolved in a tumbler of warm water. Apomorphin hypodermically administered is very certain in its effects, but gives rise to a good deal of depression and hence should be used with caution. The hypodermic dose for the production of emesis is 1/20 to 1/10 gr. (0.003 to 0.006 gm.), and it usually acts within five minutes.

When the acuteness of an attack of bronchitis has passed away and the fever is more or less gone, occasionally the cough remains trying and the sputum thick and difficult to get rid of. In such cases *potassium iodid* in small doses thrice daily is valuable.

Ammonium chlorid is also here much used. The dose is 5 to 20 gr. (0.3 to 1.3 gm.), and the unpleasant taste may be partially covered with the liquid extract of licorice, or it may be given in a mixture somewhat as suggested in Squire's Companion to the British Pharmacopœia.

℞ Ammonii chloridi	dr. ii	8.00 gm.
Syrupi limonis	oz. ½	15.00 c.c.
Spiritus chloroformi	dr. i	4.00 c.c.
Aquæ	ad oz. vi	200.00 c.c.

Misce, fiat mistura.

Sig. A tablespoonful every two to four hours.

Lozenges, containing 2 gr. (0.3 gm.) of the drug, are useful.

Potassium bichromate has been recommended by E. G. Paxton and others at this stage of the disease, where the sputum is thick and tenacious and hard to bring up. The usual dose is 1/64 gr. (0.001 gm.) every hour or two.

On the other hand, in some cases, late in the disease, the sputum remains or becomes very profuse. Here the *mineral acids,* especially the nitro-hydrochloric acid in doses of 5 to 20 minims (0.3 to 1.3 c.c.), appear, in some obscure way, to lessen this excess. Most hospitals have an acid expectorant mixture for this purpose, such as the one at St. Bartholomew's, which is as follows:

℞ Acidi nitrohydrochlorici diluti	min. x	0.60
Tincturæ aurantii	min. xx	1.25
Aquæ chloroformi	ad oz. i	30.00

Misce, fiat mistura.

Sig. Thrice daily after meals.

With it may be combined 5 to 10 drops of tincture of *nux vomica.*

After the fever and all acuteness have subsided, the patient should be out of bed and in the open; a change of air, especially to the seaside, is now of value. He will probably require some tonic, most commonly cod-liver oil or iron, or the two combined.

CHRONIC BRONCHITIS

Chronic bronchitis is a very common disease, and exists in all degrees—from a slight tendency to catarrh to one so severe that it and its complications frequently prove fatal.

The indications for treatment are:

1. To remove, as far as possible, the cause of the primary condition and of the intercurrent exacerbations.
2. To raise the patient's general health in every possible way.
3. To use means to regulate the amount of the expectoration.
4. To relieve all unnecessary cough, which cough not only tends to keep up the catarrh, but also increases the emphysema, may dilate the bronchi, and further tends to wear out the patient by causing sleepless nights and disturbed days.

Environment.—A patient suffering from chronic bronchitis should be guarded from inclement weather. These patients are nearly always better and, indeed, often apparently well in summer, and it is frequently a good plan to advise that they, if possible, spend one or two winters in the south. By so doing and thus avoiding the acute exacerbations, which are so apt to occur in winter, they may completely recover and in time once more face a northern climate with impunity. The same winter resort will not suit every case. Generally speaking, if the case be one in which the expectoration is scanty and the cough harassing, a mild and humid atmosphere will be indicated, such as is found in Florida, Nassau, Bermuda, and the West Indies, on this side of the continent, and near the coast of southern California on the west. If, on the other hand, the expectoration be profuse, a dry air is indicated, such as is found in the inland parts of southern California, Colorado, Texas, Mexico, and Egypt. When a patient has "gone South" for the winter it is most important that he does not return to his northern home too soon in the spring. One often sees this error committed with the direst results.

Watering places are much used, especially abroad, in the treatment of chronic bronchitis, and often they are valuable. One might go into great detail as to the exact composition of this or that special water, but probably, as F. A. Hoffmann, of Leipzig, says, "the mineral springs themselves are practically all of equal value. They suit human nature best, far better than pure water, and a small amount of sulphur in the water makes a pleasant and useful change. If the stronger purgatives are present, their action, of course, becomes important for corpulent patients and those having a tendency to constipation and hemorrhoids. Of course it is not a matter of indifference where we send patients, but the water itself makes little difference; the situation, the presence of forests, the freedom of the air from dust, the average amount of moisture are all of the greatest moment."

Very often the best climate for bronchitic patients is a matter of experiment, and patients will frequently try several before they find the one that suits them best. Probably the most important point is to find a place in which the patient can be the greatest number of hours in the open air, especially when this is combined with sunlight.

In the summer months a bracing climate will generally be found to be the best. The prairies of the northwest offer a beautiful inland climate, and the seacoasts on both sides of the northern part of the continent are excellent where sea air is preferred. Muskoka, the Georgian Bay and the Thousand Islands are very favorite lake and woodland resorts. In all of them the pure air and absence of dust are marked.

The great majority of patients either are not ill enough to make it expedient for them to go away for the whole winter, or, even if they are, cannot afford the time or the money to do so. In the slighter cases it is sufficient to impress upon them the necessity of avoiding night air and damp, windy weather, but the more seriously affected ones who reside in a northern country often find that they can only have comparative ease by deliberately staying indoors for some months. In elderly people this is not much of a hardship.

Cases of chronic bronchial catarrh soon find out for themselves the evil influence of "catching cold." Often, however, in endeavoring to avoid this, they defeat their end by making themselves too sensitive to change of temperature. Such people are the better for a process of "hardening" (the German *Abhärtung*). This is best accomplished by the daily use of the cold sponge, douche, or plunge, and salt water seems to be specially serviceable here. Rock salt may be added to the water to make a 1 to 3 per cent solution. Sea water contains about a quarter of a pound of solids to the gallon, and may be imitated very closely by adding common salt, 7 pounds; magnesium chlorid, 1 pound; magnesium sulphate, ½ pound, to 30 gallons of water.

According to J. H. Kellogg, cutaneous stimulation will be increased by the addition of half to one pound of chlorid of calcium to the bath. The mistake must not be made of employing the substance commonly known as chlorid of lime, the proper chemical name for which is "calcium hypochlorite." Some, such as Ortner, recommend a 5 per cent solution of salt, and certainly this is useful if the hydropathy be limited to sponging. The salt more certainly brings about a reaction than does the mere cold. By such cold applications the skin is trained to resist changes in the external temperature. Turkish baths are often useful, especially during any exacerbation. The patient, however, must take great care not to become chilled afterwards, and should quickly be got into a well warmed bed.

In the same way patients may be hardened to breathe cold air, but should be warned against mouth breathing. The nasal passages are specially adapted for warming the inspired air, as also for removing larger foreign bodies which have been inhaled. R. Hutchison reminds us that air entering the nose at freezing point is warmed to a temperature of

81° F. by its passage through the nose alone. When a chronic bronchitic must be out on a raw day it is often wise for him to wear a respirator, or at least a muffler over the mouth to prevent mouth breathing, and he should be specially cautioned against talking in the open air, which necessitates mouth breathing for the time being.

Crowded rooms, where the air is both warm and impure, should be sedulously avoided, and going into the cold air after such an exposure is specially apt to make such cases worse.

Indoors, plenty of fresh air should be provided, but draughts must be avoided; and extreme cold, such as will occur in winter if the windows be opened in the bedrooms at night, however useful it may be for tuberculous patients, is not suitable for bronchitic ones. A temperature of about 65° F. is usually the most satisfactory one for the patient, and marked variations from this, either up or down, are not advisable.

Many cases are caused, or at least made worse, by the inhalation of dust. This is seen in many occupations, such as coal mining, stone cutting, etc. Where possible, such patients should be urged to change their work for one allowing of a purer atmosphere. But many better class patients are also affected by dust, and should avoid going out on the streets on dry, windy days, and if they must do so should wear a muffler or respirator. The way in which the streets of some cities are cleaned is a constant menace to these people from the dust thus raised.

Clothing.—The clothing of bronchitic patients is an important matter. Heavy clothing is objectionable in many ways, and yet the wearers must be kept warm. Perhaps the most suitable underclothing is that made of light and porous material, of which the patient can wear two sets in cold weather. In this way the total weight of the underclothing will be less than with one heavy suit, and yet he is more completely protected from chills by the air between the two layers. The suit next the body should be of some porous material, either cotton, linen, or silk, and the outer one may be of light wool. Chest protectors are, as a rule, not advisable. When people live in a cold climate and in heated houses, it is necessary to remember that the house temperature may be almost that of summer and hence does not call for thick clothing, and yet the outside temperature may be somewhere near zero. Such a difference is best met by the use of a heavy overcoat.

Contagion.—People suffering from chronic bronchitis should avoid the proximity of individuals suffering from any acute catarrh, which is often very contagious, and acute attacks must be avoided, if possible. They are also especially susceptible to tuberculous infection and must hence avoid this as far as possible.

Tobacco.—Inhalation of tobacco smoke often excites a bronchial catarrh, and must be avoided not only by advising against the inhalation of cigarette smoke, but also by pointing out the fact that the mere breathing of an atmosphere laden with tobacco smoke from the smoking of others is almost as bad as direct inhalation.

Exercises.—Exercises, especially breathing exercises, are of great value in chronic bronchitis. It should be remembered that in these cases there is always more or less emphysema, and that hence the normal elasticity of the lungs, which in health is responsible for the expiratory act during quiet respiration, is more or less absent, which causes the expiration to become an active instead of a passive act. The result is that the excursion of the thoracic parietes is lessened, and stasis both of the respiratory tract and of the pulmonary circulation is apt to be more or less present. Regular breathing exercises, with special emphasis upon expiration, are hence valuable, and may with advantage be carried out several times a day. It is best that inspiration be done through the nose and expiration through the mouth. In this way the least possible obstruction is offered to expiration. H. E. Knopf believes that he has been successful in curing cases of chronic bronchitis by systematic exercises in deep breathing. The exercises are done before a large mirror, with the chest bare, inspiration being performed through the nose and expiration through the mouth. These exercises improve the general health in a remarkable way. Knopf urges that this deep breathing is not only the most certain expectorant, but is also the only one that is entirely harmless. The only drawback is an occasional slight giddiness, which, however, soon passes off: it is no doubt due to a mild degree of acapnia.

Compression of the lower part of the chest is often of value. It should be done during the last part of expiration, and really continues this act in a passive way, so as to make the act more ample. It may be carried out by the nurse or physician, or, as G. Hoppe-Seyler has pointed out, by the patient himself, who soon learns to do it. He is taught to place his hands on the sides of the chest, to press here at the last part of long expiration, and finally to bend forward as if bowing, which act forces the diaphragm upward and thus lessens the diameter of the chest in the vertical direction. Various forms of pneumatic apparatus have been advocated from time to time, especially abroad, but as a rule the practitioner will probably do very well without them. Postural treatment of those cases in which the bronchial secretion is excessive will be referred to later on.

When a patient with chronic bronchitis suffers an acute exacerbation he should be placed in bed, and treated as an acute case, and with even greater care than usual.

Diet and General Treatment.—Cases of chronic bronchitis are generally below weight and low in general health, and usually require to be fed up, especially with fatty foods such as milk and cream. In some cases a general dyscrasia, such as gout or uremia, may be the cause of the condition and may be best met by appropriate diet. In many of the German and Swiss spas, where chronic catarrh of the respiratory organs is treated, such as Ems, Ischl, Reichenhall, etc., the so-called "whey cure" is much used. It consists in the drinking of warm whey, either alone or mixed with mineral water, in definite quantities at fixed times. About 20 ounces (600 c.c.) are taken a day as a rule. It appears to relieve irritable laryngeal cough and to exercise a favorable influence over laryngeal and bronchial catarrh. The whey is not, of course, the exclusive diet, and it is usual to limit the amount of animal food taken and to augment the amount of fruit and vegetables. At the spas the whey is made from the milk of goats and sheep, as well as from cow's milk. I. Burney Yeo spoke highly of it. Like all spa treatments, this may be carried out at home, but is not there so successful, as all the other good influences of the spa, such as rest, good air, regular hours, etc., are apt to be missing. In children some dietetic error may be at the bottom of a persistent bronchial catarrh, and the whole régime should be carefully gone into. Habitual overfeeding or wrong feeding may be present, and other treatment will be of little avail until this is attended to.

The dietary, as presented by Robert Hutchison for dyspepsia in children, is here useful. Starch puddings—such as rice, sago, tapioca, arrowroot, and cornflour—sweets, sweet cakes, sweet fruits, jam, honey, marmalade, potatoes, turnips, carrots, should all be given in very small quantity or cut out altogether. The diet should consist chiefly of stale bread, dry toast with butter or dripping, eggs, beef, mutton, fish, chicken, bacon, tongue, green vegetables in moderation, custard, plain puddings, stewed prunes, figs, etc. The child should be permitted to eat only at meals. Tea, coffee, and other stimulants should be avoided. J. Allen calls attention to the value of this diet in the persistent bronchitis of children.

When chronic bronchitis occurs, as is not infrequent, in obese people, it usually proves very intractable. In such cases the obesity acts unfavorably, as is pointed out by Geo. A. Sutherland, in three days: (*a*) vascular obstruction occurs from increased resistance in the fatty tissues; (*b*) cardiac weakness is present from deposit of fat about the heart muscle; (*c*) pulmonary obstruction exists from excess of carbonic acid gas in the blood. It is necessary to remove the excess of fat, which is best done through three measures: (1) a lean meat diet; (2) a saline aperient every morning; (3) water to be freely drunk between meals.

Cod-Liver Oil.—Cod-liver oil, which may be here considered rather as a food than a drug, is invaluable in the common thin type of the disease, and many patients, especially children, take it during the entire winter with great benefit. G. A. Sutherland truly says, "The patient who has bronchitis every winter will probably benefit more from cod-liver oil taken throughout the cold months than from any other therapeutic measure." As a rule it is not so necessary, nor indeed so well borne by the stomach, in the summer. If there be any anemia, then it is well to combine with the oil some salt of iron. This may be done in the form of one of the numerous emulsions of cod-liver oil and iron which are on the market, or one may give the plain oil and the syrup of the iodid of iron either separately or mixed immediately before being taken. Probably the chief advantage of cod-liver oil lies in the fact of it being a fluid containing a very oxidizable fat in a very fine state of division, and also from its large vitamin content, and those preparations from which the fat has been removed are not of the same value.

In many cases it is possible, by appropriate dietetic and other treatment, to remove the underlying cause of the chronic catarrh; this is the case in gout, uremia, alcoholism, and some other toxemias. Where the heart is primarily at fault, a bronchitis will improve as this organ becomes stronger and more competent.

Vaccine Therapy.—Under removal of the cause should be considered vaccine therapy, by which the actual cause of the bronchial catarrh is attacked through the raising of the patient's specific resisting power to the special infection present. The treatment is still upon its trial, but in some long-standing cases of chronic bronchitis it has seemed to prove of great value, and it is likely that, as the technic improves, still better and more constant results will be obtained.

An autogenous vaccine is the most reliable, and where the infection is a mixed one, as is usually the case, the chief form of organism present should be made into a vaccine. A. C. Latham, who has done much work here, says that one should begin with a small dose, say one million dead bacteria. The effect should be an improvement in the patient's sensations. Any rise in temperature means that the dose has been too big. According to Latham, the remedy may equally well be given hypodermically, or by the mouth in a little normal saline on an empty and healthy stomach.

Samuel West mentions several cases of chronic bronchitis in which an almost pure culture of *pneumococci* was obtained from the sputum. A vaccine consisting of ten to thirty millions of dead pneumococci was used, but the results were not at all encouraging.

The treatment of bronchitis by vaccines is yet in its infancy, but in long-standing cases it seems to open up an encouraging field for investigation. It is, however, expert work and cannot yet be looked upon as a routine treatment.

Serum Therapy.—Serum therapy has been tried in bronchitis but without any striking results, unless indeed one include diphtheritic bronchitis, where brilliant cures have been frequently effected. G. Carrière has published a case of chronic bronchitis of streptococcic origin which was markedly improved by the use of antistreptococcic serum.

In all cases of chronic bronchial catarrh the *bowels* should be kept regular. A fractional dose of some saline every morning is a useful way of bringing this about. In plethoric cases an occasional mercurial purge is to be advised in addition to this.

Symptomatic Treatment—*Expectorants.*—The use of expectorants in chronic bronchitis has been built up entirely on empirical lines. The majority of the laity still believes that every case of cough should have a cough mixture. No doubt the profession also has tended too much in this direction, and now there is a swing of the pendulum in the other direction. Expectorants, however, hold an important place in the treatment of chronic bronchitis, and when carefully used, after a full consideration of the nature of the case, are often extremely useful in allaying the symptoms.

In the majority of cases of chronic catarrh the cough is not excessive, and if the expectoration be brought up easily, then expectorants are not required, and our efforts should be directed toward the prevention and lessening of the acute attacks and the raising of the general health by the use of tonics, proper feeding, etc., in order to bring about a more healthy condition of the bronchial mucous membrane. Where, however, the sputum is scanty and tough and the patient coughs much with little result, those expectorants which tend to make the secretion more watery are indicated. The ones that act best in chronic bronchitis are the stimulating expectorants, chiefly squills, senega and ammonium carbonate (see page 44). The iodids also act in this way and are specially indicated in gouty cases and in those cases where, along with a tough and scanty sputum, there is a tendency to wheezing and asthma. They are best given well diluted half an hour before meals, and the dose need not be more than 2 to 5 gr. Iodipin is a valuable body, and may be given with little risk of disturbing the digestion or producing other symptoms of iodism. It is said not to be split up in the stomach, but to be absorbed from the intestine. The greater part of the iodipin is oxidized in the body, and when this oxidation takes place all the iodin separates out as iodid. This action, however, is slow, and iodin may be detected in the urine for weeks after the treatment has been discontinued, according to James Burnett. The 10 per cent solution can be given in 1 or 2-dram doses mixed with a little warm milk, or the solid form may be used. This is a brown substance and may be had in tablet form.

In many patients the chief difficulty is in the morning. On first waking, the patient coughs and coughs until he brings up some tough sputum which has accumulated during the sleeping hours. He may even vomit before he succeeds in emptying the air passages. Having at last coughed up this material, he may be fairly comfortable for the rest of the day. In such cases, and they are very common, a warm alkaline aromatic draught taken on first waking usually gives great relief, by rendering the secretion more fluid and thus easily got rid of.

Such a prescription as that added to the Brompton Hospital Pharmacopœia at the suggestion of Dr. Burney Yeo may be used. It is as follows:

℞	Sodii bicarbonatis	gr. xv	1.00 gm.
	Sodii chloridi	gr. v	0.35 gm.
	Spiritus chloroformi	min. v	0.35 c.c.
	Aquæ anisi	ad oz. ½	15.00 c.c.

Misce, fiat mistura.

Sig. To be taken in a glass of hot water on first waking in the morning.

The writer has frequently found this mixture of great value.

Another useful remedy with the same object is Ems water, of which 3 or 4 ounces should be taken in hot milk on first waking; or the old-fashioned rum and milk may be used with advantage.

In cases where, during the night, the cough is dry and the sputum difficult to cough up, the alkaline mixture given above may be used with benefit at bedtime as well.

Liniments, Poultices, and Sprays.—Liniments are frequently of value in chronic bronchitis. They are the same as recommended in acute bronchitis and used in the same way. Poultices and fomentations are not required in chronic bronchitis, but during any little exacerbation a "mustard-leaf" over the upper part of the sternum often gives relief. In cases of chronic bronchitis with scanty sputum mild alkaline sprays are often of value, such as Seiler's solution.

Cough.—By the use of means, such as those indicated, to make the secretion more fluid, the cough may be so much relieved that it needs no special treatment. But frequently the cough remains excessive, and is indeed the chief symptom complained of. It may, by keeping up an irritation in the upper air passages, make the catarrh worse and a vicious circle is established—the cough causing the irritation, which in its turn causes the cough. In every case of cough it is most necessary that the practitioner, before employing any remedies for its easement, fully consider if the cough be a useful one and, if so, whether it be excessive or not. As an example of an absolutely useless cough one may cite that produced by a dry pleurisy, and as a type of a useful one that set up by the presence of a removable foreign body in the air passages. The first, of course, may be freely checked, while the latter should be encouraged or at least left alone. The same rule holds good in the coughs accompanying bronchitis. In one case a dry, irritable cough may be making the patient miserable, and yet is only doing harm and should be checked, while again another patient may be coughing only to get rid of a quantity of secretion which is threatening to asphyxiate him, and such a cough, of course, should be encouraged.

Insomnia is frequently produced by coughing, and the late Dr. Gustave Schorstein, of the London Brompton Hospital, used to say that the best hypnotic in chronic bronchitis was strychnin. It, of course, acts by helping the cough and thus clearing the tubes of secretion, the presence of which keeps the patient coughing and hence awake. It may be given in the form of the hydrochlorid or sulphate in doses of 1/60 to 1/20 gr. (0.001 to 0.003 gm.) either by the mouth or hypodermically, or nux vomica may be used in doses of 5 to 10 minims of the tincture, either by itself or added to any other medicine that the patient is taking. A useful prescription for cases with moderate secretion is as follows:

℞

Tincturæ nucis vomicæ	min. v to x	0.35-0.70 c.c.
Tincturæ scillæ	min. xx	1.25 c.c.
Ammonii carbonatis	gr. iii	0.20 c.c.
Syrupi tolutani	dr. ½	2.00 c.c.
Infusi senegæ	ad oz. ½	15.00 c.c.

Misce et fiat mistura.

Sig. Thrice daily after meals.

An old recipe which the writer has often found useful in cases having a good deal of cough and a small amount of sputum is the following:

℞ Tincturæ camphoræ compositæ
Oxymellis scillæ
Syrupi tolutani āā ℥i (30 c.c.).
Misce, fiat mistura.
Sig. A teaspoonful from three to six times daily in water.

Often the cough is largely kept up by an irritable condition of the pharynx, and in such cases barley water, flavored with lemon, is a pleasant and efficient remedy and will frequently relieve an irritating nocturnal cough, if sipped as required. In cases of this kind it is important to tell the patient not to cough if he can possibly help it, the cough keeping up the irritation. Such advice is on a parallel with telling an individual with an itchy eczema not to scratch. In both cases many unnecessary irritations of the inflamed parts may thus, by the exercise of the will, be stopped.

In cases with much irritation of the upper air passages, sprays, as already mentioned, are useful. Also inhalations from the handkerchief or through a Burney Yeo or some other form of inhaler. Ten drops of a 20 per cent solution of menthol in alcohol poured on the sponge and used for hours may give much relief. Or the following:

℞ Menthol	1 part
Eucalyptol	2 parts
Benzoinol	3 parts

Misce, fiat mistura.

Sig. Inhale from an inhaler or handkerchief.

As a rule, opium is contra-indicated in chronic bronchitis, but in these irritable cases it is often of value and may be used cautiously in small amounts. Paregoric, compound ipecacuanha powder, and pill of ipecacuanha with squills are good preparations here. A useful prescription for controlling unnecessary coughing is the following:

℞

Liquoris morphinæ hydrochloridi	dr. i	4.00 c.c.
Acidi hydrocyanici diluti	dr. ½	2.00 c.c.
Syrupi pruni virginianæ	oz. ½	15.00 c.c.
Infusi rosæ acidi	ad oz. iii	100.00 c.c.

Misce et fiat mistura.

Sig. A teaspoonful without water as often as every four hours.

This forms a mixture which is very soothing in an irritable cough. Its action seems to be partly local and hence is best marked when the medicine is taken undiluted. As a rule, however, in such chronic coughs as we are discussing, the use of sedative cough mixtures, if employed at all, should be reserved for the night.

It is scarcely necessary to say that in a chronic condition like the one under discussion, opiates in any form should be avoided as largely as possible, and if a prescription containing such is for a time considered necessary it should be marked "not to be repeated," in order to avoid the possibility of the patient acquiring the opium habit.

Profuse Expectoration.—With very profuse expectoration the medicinal treatment is quite different. Here the cough is life-saving and nothing must be done to check it; but, on the contrary, if the patient be weak and the muscular power low, it should be encouraged. This can be best done by the use of strychnin. Our efforts should be directed toward lessening the amount of secretion, both by raising the general tone of the patient by the use of tonics, good food, etc., and more directly by the use of certain drugs which have the power of directly lessening the abnormal secretion. Such drugs are belladonna, the mineral acids, and the so-called aromatic expectorants.

Belladonna acts powerfully as a drier-up of secretion, and also as a respiratory stimulant, but must be used with care as it tends also to dry up the saliva and other secretions, and may give rise to discomfort in consequence. Its active principle, atropin, is a very powerful stimulant of the respiratory center; it also lessens the respiratory reflex, dulling the terminations of the vagus, and thus tends to ease the cough; but this dulling action is not so marked as it is in the case of opium, and the other contra-indications are not present. The tincture of belladonna may be given in doses of 10 minims (0.6 c.c.) thrice daily, and will certainly lessen the amount of secretion. Sometimes, however, it will be found that, although the secretion be lessened, the patient is not so comfortable.

The *mineral acids,* especially the nitro-hydrochloric acid, are valuable in lessening expectoration, as in the later stages of acute bronchitis.

The *aromatic expectorants,* such as *creosote* and *terebene,* tend to lessen bronchial secretion. They are specially indicated where any fetor exists in the secretion, and will be referred to later.

When the bronchial secretion is excessive, *posture* is an important factor in the treatment of the condition. As is well known, in health the cilia of the epithelial cells lining the respiratory tract are constantly tending by their movements to bring the normal secretion, and with it any particles of dust which may have alighted upon it, toward the outlet of the respiratory tract, where it is swallowed or expectorated. But, when the epithelium is changed, as it is in chronic catarrh, and the cilia no longer are there thus to move the secretion onward, this material, often excessive in amount, collects in the air passages and must all be coughed up. If patients with excessive secretion can be made to sleep with the foot of the bed a little raised, gravity helps, or at least no longer retards, the passage of this secretion toward the outlet. F. Forchheimer specially emphasized the value of this postural treatment. The foot of the bed should not be raised more than two or three inches at first, but gradually can be put up to double that height. Beds are now made by various manufacturers, the ends of which can be conveniently raised or lowered to any desired degree by means of levers acting on cogs.

In rare instances the expectoration in cases of chronic bronchitis becomes *fetid.* In such instances there is always a suspicion of actual dilatation of the bronchi, which may be marked enough to merit the term of bronchiectasis. In fetid cases the aromatic expectorants are indicated.

Aromatic Expectorants.—*Creosote* is much used, and is best given in pearls or capsules containing 2 or 3 minims in each. Or it may be given as the *mistura creosoti* of the B. P., or combined with cod-liver oil. *Guaiacol carbonate,* a derivative of creosote, is a useful expectorant here, and if given in doses of 5 to 10 gr. thrice daily or every four hours (in capsule form), it will soon show its characteristic odor in the breath. *Tar,* in doses of 5 to 10 minims (0.3 to 0.6 c.c.) in capsules, may be given with the same object.

Turpentine may also be used in such cases, and can be given in capsules containing 5 to 10 drops, or in a mixture as follows:

℞	Olei terebinthinæ	dr. i	4.00 c.c.
	Mucilaginis acaciæ	oz. ii	60.00 c.c.
	Emulsionis amygdalæ	ad oz. vi	200.00 c.c.
	Misce, fiat mistura.		

Sig. A tablespoonful thrice daily after food.

Terebene is another aromatic which is employed in such cases. Its odor is more pleasant than that of turpentine, which it otherwise resembles.

It is given in doses of 5 to 15 drops, conveniently dropped upon lump sugar, or it may be dispensed in capsules, or as a draught, such as that of the Victoria Park Hospital, which is as follows:

℞	Terebeni	min. x	0.60 c.c.
	Mucalaginis tragacanthæ		
	Glycerini	āā dr. i	4.00 c.c.
	Aquæ cinnamoni	ad oz. i	30.00 c.c.

Misce, fiat mistura.
Sig. To be taken thrice daily after meals.

Or the aromatics may be inhaled, and thus more directly applied to the diseased mucous membrane. Such a mixture as this is useful:

℞ Terebeni
Acidi carbolici liquifacti
Spiritus chloroformi āā ʒij (8.00 c.c.).
Sig. To be inhaled from a Burney Yeo inhaler for hours daily.

Or one as recommended by J. C. Briscoe:

℞	Creosoti	dr. iii	12.00 c.c.
	Thymol	dr. ii	8.00 gm.
	Acidi carbolici liquifacti	dr. i	4.00 c.c.
	Spiritus chloroformi	ad oz. i	30.00 c.c.

Misce et fiat mistura.
Sig. To be inhaled frequently.

Or one may use the inhalation containing menthol and eucalyptol in benzoinol already given (page 56).

Various sprays may also be employed here, such as one of Seiler's solution, or one of ipecacuanha wine, as recommended by Ringer and Murrell.

Among the newer and less commonly used methods of treatment of chronic bronchitis one may mention the following:

X-ray.—The X-rays have been tried in chronic bronchitis, and some good reports of the results have been published. Thus Dr. Schilling of Nuremberg at the German Congress of Medicine in 1906 stated that he had treated a number of patients in this way. With one exception the results were favorable and the bronchial secretion decreased. Even in cases of months' duration the expectoration became less copious, and in some instances totally disappeared without any undesirable complications. The general health of the patients was improved and their respiratory difficulties were lessened. One would, of course, like to know more about these patients—for example, whether any of them were of years' duration, as many a subacute bronchitis lasting a few months clears up completely under almost any treatment.

Incandescent Light Baths.—A. von Strümpell strongly advises the use of Kellogg's incandescent light baths in chronic bronchitis. In his hands the treatment seemed to be very successful. It is specially advised in the dry form of the disease. The light produces hyperemia of the skin and sweating, and should have some counterirritating effect, if nothing more.

Other Remedies.—As has been already said more than once, cases of chronic bronchitis are of all degrees, from those that are so slight as to be scarcely considered ill to those who are more or less completely invalided. These severe cases tend to get gradually worse in spite of all treatment, and, eventually, from increasing emphysema and failing right heart, become completely invalided. In such cases, where the dyspnea is marked and there is more or less cyanosis, the inhalation of oxygen is of some temporary value. The gas should be bubbled through water at the rate of about 120 bubbles per minute, and inhaled through a rubber tube placed in a nostril, or by means of a special inhaler such as the Meltzer.

Strychnin hypodermically helps these sufferers very much and should be freely given. Alcohol, especially good brandy, is often valuable, particularly in the aged. The heart must be supported in every way possible in these advanced cases, and digitalis is usually given freely and with some benefit occasionally.

When cyanosis is marked, *bleeding* helps to keep the patient going a little longer sometimes, and may be repeated at intervals of a few weeks with temporary benefit on each occasion.

PLASTIC BRONCHITIS

Plastic bronchitis is a rare disease, the essential nature of which is far from clear. It is characterized by the periodical formation in the bronchial tubes of a fibrinous material, which, after causing much distress from dyspnea, is thrown off and expectorated in a more or less intact condition as casts.

Treatment.—So little is known about the essential nature of this disease that we can give no general directions to our patients, beyond those bearing upon the improvement of the general health. No diet has any perceptible effect upon the condition.

When an attack occurs, the one drug that has proved of undoubted value is the iodid of potassium; it no doubt acts by causing a pouring out of thin bronchial secretion, which helps to loosen the adherent casts. It should be given in repeated doses of say 2 to 5 gr. every four hours. These small doses have a more marked tendency to produce iodism than have the larger ones, and this is what we want here.

Pilocarpin acts in the same way, and may be given by the mouth in expectorant doses of 1/20 to 1/10 gr. (0.003 to 0.006 gm.) of the nitrate or hydrochlorid every four hours.

Apomorphin also acts as a producer of freer secretion, and may be given in small doses for this purpose. Doses of 1/32 to 1/10 gr. (0.002 to 0.006 gm.) of the hydrochlorid given by the mouth every two to three hours will soon produce an abundant secretion. The emetic action of this drug is not nearly so well marked when it is given by the mouth as when given hypodermically; on the other hand, the expectorant action is better developed, and hence it should be given in this way in this disease.

In severe cases it may be advisable to use an *emetic,* with the double object of producing a free secretion from the bronchial mucous membrane, which always accompanies nausea; and also to produce vomiting, which may at the same time expel the contents of the bronchi.

Ipecacuanha in 20-gr. (1.3 gm.) doses will act well here; or perhaps, better still, a hypodermic injection of 1/15 to 1/10 gr. (0.004 to 0.006 gm.) of apomorphin. When, however, the patient is very distressed and cyanosed, it becomes a nice question to judge whether one should or should not run the risk of heart failure from the use of an emetic. In a case seen with the late Dr. R. J. Dwyer the patient was so near death from asphyxia that we feared to put any further strain on the heart already much dilated. However, after the patient had become almost black from cyanosis, pulseless, and apparently practically dead, she herself vomited and soon brought up a quantity of sputum containing an almost complete cast of the respiratory tract below the trachea, and then completely recovered. In a similar attack, which occurred some months later, she died before skilled assistance was obtained.

The fact that the casts are soluble in limewater led to the suggestion by Biermer that an inhalation of atomized limewater be used, and a case of its successful employment was reported by Waldenburg. No recent reports of its use have been made. Inhalations of creosote vapor have been suggested by Fowler and might be tried. It can be conveniently inhaled from a Burney Yeo zinc inhaler, and if too irritating may be mixed with equal parts of spirit of chloroform.

Morphia is, of all drugs, the one most contra-indicated here and should never be used to relieve the distress. It dries up the secretions, lessens the lung reflex, and tends to weaken the respiratory center. In one case within the writer's knowledge it seemed on several occasions to precipitate an attack.

BRONCHIECTASIS

Environment.—It is most essential that the patient's general health should be kept up by abundance of pure fresh air and plenty of nourishing food. This is the most important part of the treatment as regards environment.

> "Not drugs, but a suitable place of abode and appropriate occupation are the important factors in the treatment of the disease. The patient must above all things avoid the haunts of men; crowds are hotbeds where the infections most to be dreaded multiply" (Hoffmann).

A dry, bracing climate is the best here, such as is found in southern California, on the tablelands of South Africa, or the high elevations on islands in the West Indies. Egypt is a good place. The prairies of the northwest of this continent are eminently well adapted for these cases. The air there is dry and pure to such an extent that dead things such as game tend to dry up rather than decay, and the constant inhalation of such air may do much to lessen the putrefaction in the cavities. It is important to remember, when advising patients as to traveling, that, while the fetor exists to any marked extent, it is difficult for them to travel with comfort on this account. In choosing a climate it is most necessary to find one where the patient may be the greatest possible number of hours in the open air and sunshine.

Chills and dampness are to be avoided, as they tend to bronchitis; and this is a bad thing in these cases in many ways. Whenever there is any fever the patient is best in bed, and slight intercurrent febrile attacks are very common.

Warm sea water bathing, or at least salt baths followed by a cold douche and a brisk rubdown, are valuable. Exercises, especially those calling for slow and ample breathing, are serviceable in expanding the chest and improving the general circulation. Special exercises, undertaken with the object of emptying the bronchiectatic cavities, are referred to later on.

Diet.—In many cases there is much loss of weight, which calls for as liberal a diet as the patient can assimilate (see Chronic Bronchitis).

Specific Treatment.—No medicine or other remedy can, of course, directly restore the caliber of the diseased bronchial tubes, but in acute cases, such as occur chiefly in children, there still remains a tendency to such return to the normal, which may be aided by remedies which lessen the amount of the infection of the sputum and relieve the strain of coughing, and especially by every measure which tends to the restoration of the general health. Where the infection is chiefly a single one, something may be hoped for from vaccine therapy.

It is most important to empty the cavity frequently, and the patient soon finds the posture in which this most easily occurs. If he leans forward, or assumes some other position, a cough comes on and he brings up more or less of the contents of the cavity; the altered position has caused some of the secretion to gravitate into a more normal part of the bronchus, and the reflex cough then set up does the rest. A. Quincke, William Ewart, and others have developed this idea of posture in great detail and have elaborated methods for both the occasional and the continuous emptying of the cavities. G. Hoppe-Seyler also lays great stress upon the importance of posture and proper exercises, and urges that in every case of the disease, but more especially in the aged, every effort should be made to ensure complete emptying of the cavities and air passages. Benefit may be derived from breathing exercises supplemented by compression of the lower part of the chest during expiration. In sacculated bronchiectasis of the lower lobes it is difficult to evacuate the secretion, but this may be aided by placing the patient in the Trendelenburg position, and he may train himself to recline in this position for an hour or two night and morning.

Surgical Treatment.—In some cases, where the bronchiectatic cavity can be clearly localized and appears to be single, surgical interference has proved of value and has brought about a cure, which could scarcely have been hoped for otherwise. The cavity is opened and drained through the parietes. One or more ribs should be resected so that the walls of the cavity can collapse and healing finally take place. Such treatment should not be lightly undertaken. In the first place, the localizing of the cavity is far from easy and, even when it has seemed to be quite definite, the cavity has proved to be only one of several, some of which could not be reached. This is notably the case in children, as was shown some years ago by John Thompson, of Edinburgh. F. A. Hoffmann had collected, up to 1904, records of 23 cases operated upon, with 11 recoveries and 12 deaths. Hofmockel, in 80 cases operated upon for abscess, gangrene, and bronchiectasis, says the best effects were obtained in abscess and the worst in bronchiectasis. Roswell Park reported, some years ago, 23 cases operated upon, with 9 deaths. He pointed out that in the absence of pleuritic adhesions the operation is difficult and dangerous; otherwise it is easy, especially if the cavity be superficial.[2]

For the present one may say that only exceptional cases should be operated upon. Those following definite fibrosis of the lung are more likely to be single than are those complicating bronchitis, and are hence the ones most likely to be benefited by operation. Samuel Robinson says here:

> "If the cavity is bronchiectatic, it should almost never be drained. A bronchiectatic fistula and chronic sinus may persist. Numerous smaller cavities in the immediate neighborhood of the one drained continue to secrete profusely. The disease continues its course. . . . The surgery

---

[2] Excision of the lobe of the lung containing the cavity, or cavities, has now been done many times and is the surgical method most to be recommended.—Editor.

of bronchiectasis consists in two possibilities: immobilization of the lung by nitrogen gas injections into the pleura, or resection of the ribs."

Forlanini, according to Robinson, reports two complete cures by the nitrogen injection treatment, and it would seem to be an excellent thing to try, as it has been used so successfully in cases of pulmonary tuberculosis with cavities, and is practically free from risk. In one case in which the patient had a hectic fever, profuse sweating, marked emaciation, with about 300 c.c. daily of most foul sputum, the writer gave great relief by artificial pneumothorax. The temperature became normal, the sputum almost ceased and the patient gained 7 pounds a week, and a year later wrote that he was practically well. The presence of pleural adhesions will often make this method of treatment impracticable, it being impossible to collapse the lung.

Symptomatic Treatment.—The symptoms which call for treatment are chiefly (*a*) emaciation, (*b*) cough, (*c*) profuse expectoration, (*d*) fetor.

Emaciation.—See Diet.

Cough.—The cough is a useful one and seldom needs to be directly treated, much less checked. It will, as a rule, be a grave therapeutic error to administer opium or its derivatives with the object of lessening it. But often, in addition to the bronchiectasis, there exists a more or less generalized catarrh of the respiratory tract, which keeps up a frequent and (partly at least) useless cough, and in such cases soothing remedies (even containing opium in small amounts) are indicated.

Fetor.—The most reliable remedy for lessening the fetor of the sputum is creosote. The drug may be inhaled in various ways, or it may be given internally.

Creosote Bath.—The creosote vapor bath, as originally suggested by Arnold Chaplin, is probably the most efficacious medicinal treatment of bronchiectasis known. The details of this method, as given by Godlee and Fowler, are as follows:

A small room should be set apart for the purpose, and should be cleared of furniture, except such articles as wooden chairs and a table. In order to prevent the vapor of the creosote from penetrating them, the patient should have over his clothes a garment with sleeves shaped somewhat like a smock frock. The eyes should be protected by well-fitting goggles similar to those worn by Alpine climbers when on the snow, or a mask may be worn, made of surgical strapping, with two watch glasses between the adherent surfaces corresponding in position with the eyes, and tied with tapes at the back of the head. Plugs of cotton wool should be inserted in the nostrils. Women should cover the hair with a cap similar to that used when bathing. Ordinary commercial creosote should be poured into a metal saucer on a ringed iron tripod, and the saucer heated by a spirit lamp placed under it. If the floor is of wood, the tripod should be placed upon a large flat stone slab, as the creosote may run over the edge of the saucer. As the creosote is heated dense fumes rise and fill the room. The effect upon the patient is to produce violent cough attended with profuse expectoration, nearly two-thirds of the daily amount being often expectorated during a bath. Vomiting may follow the cough. The process is certainly not a pleasant one, but it is remarkable how quickly the patients become accustomed to it. They usually state that the breathing is much freer after the bath, and that they enjoy a much longer interval of almost complete freedom from cough and expectoration. At first the bath may be given on alternate days for fifteen to twenty minutes, but when the patient has become accustomed to the treatment the bath may be given daily, and the duration gradually increased to an hour or an hour and a half. In severe cases it may be well to give the bath twice daily. Under such treatment the fetor may disappear within a short time, but this does not always follow. In some cases the quantity of expectoration greatly decreases; in others it is little affected. The treatment should be persisted in for several months. There is often a marked increase in weight under its use.

Short of the creosote bath, great benefit may accrue from inhalations of creosote from some special inhaler, such as that recommended by Burney Yeo. A. McPhedran speaks of a case which was successfully treated by the inhalation of creosote from a nebulizer operated by compressed air. The case followed influenza.

Many other aromatic inhalations may be used with the same object, especially menthol and eucalyptus, as in the following:

℞	Menthol	1 part
	Eucalyptol	2 parts
	Benzoinol	3 parts

Misce, fiat mistura.

Sig. To be inhaled from a handkerchief or from an inhaler.

Oxygen.—W. H. Herringham suggests the inhalation of oxygen from a Dr. George Stoker's inhaler. It should be used for six hours daily. Great improvement may result. He describes a case in which this treatment was alternated with the creosote vapor bath, and the patient improved much more, both as regards the fetor and the quantity of expectoration, under the oxygen than under the creosote. A point in favor of the oxygen is that it is much less unpleasant than is the creosote. The best way of using oxygen is by the oxygen chamber with which some hospitals are now provided, but it can be quite well given through a soft catheter placed in one nostril. These various inhalations should be used just after the patient has more or less emptied his air passages by coughing.

Expectorants.—Taken internally the aromatic expectorants have some action in lessening the fetor and diminishing the quantity of expectoration.

Creosote, terebene, and guaiacol carbonate are the ones chiefly used here.

Creosote should be given in small doses, gradually increased as the patient can stand it. It may be given in capsule form or in a mixture, and is often well combined with cod-liver oil. Two drops thrice daily is a sufficient initial dose.

Terebene is well given in capsules containing 5 to 10 drops, or it may be administered as the *haustus* of the Victoria Park Hospital. This is made up as follows:

℞	Terebeni	10 min.	0.65 c.c.
	Mucilaginis tragacanthæ		
	Glycerini	āā 1 dr.	4.00 c.c.
	Aquæ cinnamomi	ad 1 oz.	30.00 c.c.
	Misce, fiat mistura.		

Sig. Thrice daily after meals.

The *guaiacol carbonate* should be given in 5 to 10-gr. doses (0.35 to 0.65 gm.) thrice daily, or even every four hours, and will soon make itself evident in the breath.

Vivian Poore suggested the use of garlic and reported very favorably upon it. A clove of garlic is chopped up and mixed with beef tea or given in capsules. The *essential oil of garlic* (*oil of allyl*) may be given in drop doses in capsules thrice daily instead, and soon causes the characteristic odor in the breath. *Myrtol* has been advocated in the treatment of putrid bronchitis and bronchiectasis. It is a volatile oil obtained from the leaves of the common myrtle, and when taken internally is eliminated chiefly by the respiratory tract, and acts as an antiseptic and deodorant. It may be given in capsules containing 2 minims (0.12 c.c.) taken frequently. If the sputum be thick and tenacious and is only brought up with difficulty, iodid of potassium is of value. It should be given in doses of 3 to 5 gr. (0.2 to 0.3 gm.) thrice daily. An alkaline aromatic draught taken first thing in the morning is useful in thinning the sputum and thus allowing of it being got rid of more easily. Such a one as that mentioned under Chronic Bronchitis is a good one.

Injections.—Intratracheal injections have been much employed in this condition. They were first introduced by A. Rosenburg for tuberculosis of the lungs and larynx. In 1886 Sehrwald and Reichert, by experiments on dogs, calves, and sheep showed that fluids so injected made their way into the finest bronchioles. Sehrwald's conclusions were: (1) that large quantities of fluid could be injected without discomfort; (2) that the fluids not only passed into the alveoli, permeating the surrounding structures, but also reached the peribronchial and pleural fibrous structures and even penetrated the cartilages; (3) that the lungs absorb more actively than does the digestive tract, or even the subcutaneous tissues, the rapidity of absorption depending upon the extent of the absorbing surface —so much so, indeed, that the lung of a dog can absorb four times its own weight in less than five days; (4) that medicines thus introduced act in smaller doses and more rapidly than when introduced in any other way.

A formula frequently used for injection is the following:

℞ Menthol	10 parts
Guaiacol	2 parts
Olive oil	88 parts

A dram to be injected.

Colin Campbell improved the method by employing Price's distilled glycerin instead of olive oil. Other glycerins are too irritating for use. Menthol and guaiacol have been most used in this way, but more recently Campbell has substituted *izal.* He generally empties a syringe holding 100 minims (6.5 c.c.) at one squirt and repeats this two or three times at each sitting. As much as 3 ounces (100 c.c) may be given in a day. Colin Campbell's method, as quoted by William Ewart, is as follows:

"The tube should be passed rapidly into the larynx, and should fit the curve at the base of the tongue and lie lightly against it, thus fixing the epiglottis and preventing spasm. The squirt should be delivered like lightning—either with inspiration, or during a slightly prolonged interval following expiration. If the operation be properly performed, the taste of the fluid injected should not be perceived by the patient," neither is there cough, pain, or asphyxia. The daily use of such injections may produce a great improvement in the degree of fetor and the quantity of expectoration.

BRONCHIAL ADENITIS

Inflammation of the bronchial glands, as a sequel to acute infections of the lungs and bronchi, is very common. It is specially apt to occur in children as a complication of bronchopneumonia and bronchitis. It is also an almost constant accompaniment of whooping-cough; in fact, the spasms in this disease have been attributed to the enlargement of the glands, although with little evidence. In measles and influenza the condition is common. The adenitis complicating these acute infections as a rule clears up completely in time and does not call for any special treatment. But in the tuberculosis of children involvement of the bronchial glands is extremely common and may be of serious and even fatal import.

Therapy.—The treatment of the latter is largely that of tuberculosis in general, and may be summed up as the placing of the patient in the best possible environment for the raising of his general health and consequent resisting power. Fresh air and abundance of good and nourishing food are essential. Residence at the seaside is of the greatest and almost specific value in this condition of tuberculous adenitis and should be insisted on whenever possible. The sea air, in some not clearly understood way, helps these patients to a very marked degree. Adolphe d'Espine, of Geneva, published several years ago a list of 119 cases of recognizable enlargement of the bronchial glands, all treated with eight months' residence at the seaside, with a cure both local and general in 55 cases, that is, 46 per cent. In 54 children the physical signs of enlargement remained, but the general health, often seriously impaired, had been reëstablished. The value of sea air has also recently been emphasized by L. W. Ely, the heading of his article itself being suggestive, namely, "Treatment of Cervical Tuberculous Adenitis at *Sea Breeze Hospital.*" (The italics are ours). Rollier speaks enthusiastically on the curative effect of heliotherapy in these cases. X-ray treatment has also proved of value.

Iron, especially in the form of the syrup of the iodid, is generally useful, and cod-liver oil is always indicated during the cold months, when it can be taken. It will be found that where a child cannot take emulsions he will take the pure oil quite well. There often seems to be a craving in these tuberculous children for fat, which the oil appears to meet.

Tuberculin finds a very hopeful field in these cases of adenitis, and a course of it is the best thing possible in the way of active interference.

Commencing with a very small dose of the old tuberculin, say 1/20,000 mg., it should be administered once a week, and as a rule the dose should be doubled each time. If there be any local or general reaction, then the dose is probably too large, although it should be said here that recently some authorities are advocating these larger doses. Care should be taken not to give a further injection while any reaction is present. There are now many tuberculins each having its advocates. The course of treatment should last for several months. Carmalt Jones recently gives the results of this treatment, in tuberculous glands generally, as carried out at the Westminster Hospital, London. There were 79 cases treated, and of these 27 were cured, 22 much improved, 18 better, 8 unchanged, and 4 worse. He says that "in any average 8 cases, in which inoculation is adopted (with correct dosage), 5 will probably show marked improvement, of whom 2 or 3 will be cured; 2 will probably improve slightly, and 1 or 2 will fail. The best results are in young children and in young adults between fifteen and twenty-five. The worst results come between ten and fifteen years."

As a rule, the tuberculin should be given hypodermically, although some workers, for example, A. C. Latham, think that it acts as well when given by the mouth and S. Solis-Cohen confirms this.

The bovine type of the disease, to which class these cases mostly belong, seems specially amenable to the human type of tuberculin. This point is brought out by Nathan Raw. He commences with 1/10,000 mg. of Koch's old tuberculin and doubles the dose each week until 1/10 mg. is reached, beyond which he never goes. If the case requires it, he begins a second course, after a short interval, this time commencing with a larger dose and therefore more quickly reaching the maximum. Under the tuberculin treatment, which, of course, should only accompany all other necessary therapy, cervical glands may be seen to improve very rapidly and the less easily observed bronchial ones may be presumed to follow the same course.

REFERENCES

Allan, J. Persistent Bronchitis in Children, Practitioner, April, 1910.

Barlow, W. Jarvis. Definition of Climatic Therapeutics, Hare's Modern Treatment, i, 156.

Briscoe, J. C. Practitioner, October, 1909.

Burnett, James. Therapeutic Action of the Iodids, Lancet, ii, 648, 1906.

Campbell, Colin. The Treatment of Respiratory Affections by Large Med. Injections through the Larynx, Med. Chir. Tr., 93, 1895.

Campbell, Harry. Clin. Journ., Jan. 5, 1910.

Carrière, G. Bronchite Chronique Streptococcique, Efficacité du Sérum Antistreptococcique, Presse méd., June 5, 1897.

Chaplin, Arnold. Brit. Med. Journ., i, 1371, 1895.

Claisse, P. Bronchite in Maladies des Bronches et des Poumons, 53, 1910.

Cushny, A. R. Pharmacology and Therapeutics, 217, 282,

Davis, N. S. Belladonna Inhalation in Acute Bronchitis, Brit. Med. Journ., i, 542, 1886.

D'Espine, Adolphe. La Cure Marine de la Scrofule à l'asile de Cannes, Arch. de Méd., Dec., 1904.

Dixon, W. E. Facts and Fancies in Pharmacology, Brit. Med. Journ., ii, 539, 1909.

Dugan, W. J. Handbook of Electro-Therapeutics, 135.

Dunbar, W. P. Zur Ursache und Specifischen Heilung des Heufiebers, Deutsche med. Wchnschr., 1903.

Ely, L. W. Treatment of Cervical Tuberculous Adenitis at the Sea Breeze Hospital, Am. Journ. Obst., 1910.

Ewart, Wm. Bronchitis, C. Allbutt's System of Medicine, v, 114.

——— Bronchiectasis, Ibid., 152.

Fagge, J. Hilton. Principles and Practice of Medicine, ii, 295.

Febray, Paul. Pest. med.-chir. Presse, 1907. Quoted in Gaz. méd. de Par., 1907.

Fetterolf, Geo. Hare's Modern Treatment, ii, 385.

Forchheimer, F. Treatment of Chronic Bronchitis, Am. Journ. Med. Sc., 1909.

——— The Prophylaxis and Treatment of Disease, Ibid., 354.

Fowler and Godlee. Diseases of the Lungs, 97, 122.

Friedenwald and Ruhräh. Diet in Health and Disease, 433.

Gibson, G. A. Varieties and Treatment of Asthma, Lancet, ii, 368, 1911.

Gillette, H. F. Untoward Results with Diphtheritic Antitoxin, Journ. Am. Med. Ass., Feb. 13, 1909.

Goodhart and Spriggs. Asthma and Hay Fever, C. Allbutt's System of Med., v, 46.

Henderson, V. E. Expectorants, Journ. Pharmacol. & Exper. Therap., Oct., 1910.

Herringham, W. H. Lancet, i, 1177, 1909.

Hoffmann, F. A. Nothnagel's Encycl. Pract. Med., Dis. of Respiration, 137, 207.

Holt, L. Emmett. The Bacteriology of Acute Respiratory Infections in Children, As Determined by Cultures from Bronchial Secretion, Journ. Am. Med. Ass., 1910.

Hoppe-Seyler, G. Deutsche med. Wchnschr., July, 1909.

Hutchison, R. Applied Physiology, 178.

Jagic, N. V. Berl. klin. Wchnschr, 583, 1909.

Jones, Carmalt. Introduction to Ther. Inoculation, 134.

Kayser, C. Therap. Monatsh., Berl., No. 3, xxvi, March, 1911.

Kellogg, J. H. Rational Hydropathy, 955.

Knopf, H. E. Berl. klin. Wchnschr., June 15, 1908.

Latham, A. C. Practitioner, Dec., 1910.

Lemann, I. I. Am. Journ. Med. Sc., Dec., 1911.

Lloyd, Wm. Asthma, Its Causation and Treatment, Brit. Med. Journ., i, 143, 1909.

Lord, F. T. Boston Med. & Surg. Journ., 662, 1902.

——— Tr. Ass. Am. Physicians, 1910.

Matthews, C. The Use of Adrenalin in Acute Asthma, Brit. Med. Journ., i, 441, 1910.

McPhedran, A. Disease of the Bronchi, Osler's Mod. Med., iii, 641, 696.

Melland, B. The Treatment of Spasmodic Asthma by Hypodermic Injections of Adrenalin, Lancet, i, 1407, 1910.

Meltzer, S. J. Bronchial Asthma, Tr. Ass. Am. Physicians, 1910.

Moon, R. O. Folia Therap., July, 1910.

Murrell, Wm. Brit. Med. Journ., ii, 1611, 1886.

Newlin, A. Bronchitis, Hare's Modern Treatment, ii, 407, 417.

Noon, L. Prophylactic Inoculation against Hay Fever, Lancet, i, 1572, 1911.

Ortner, N. Treatment of Internal Disease, 288, 295, 297.

Osler, Wm. Practice of Medicine, 53.

Park, Roswell. Ann. Surg., 1887.

Paxton, E. G. Am. Journ. Clin. Med., Jan., 1908.

Poore, G. Vivian. Nervous Affections of the Hand and Other Clinical Studies, 1897.

Quincke, A. Zur Behandlung der Bronchitis, Berl. klin. Wchnschr, xxxv, 525, 1898.

Rapin, Dr. Rev. méd. de la Suisse Rom., April, 1911. Quoted from Epitome, Brit. Med. Journ., ii, 16, 1911.

Ringer, Sydney. Brit. Med. Journ., ii, 1543, 1896.

Robinson, Samuel. Boston Med. & Surg. Journ., Sept. 14, 1911.

Rudolph, Robert D. Am. Journ. Med. Sc., Dec., 1912.

Sawyer, Sir James. Folia Therap., 35, 1907.

Schäfer, F. München. med. Wchnschr., July, 1907.

Schilling, Dr. Lancet, i, 1777, 1906.

Sehrwald and Reichert. The Percutaneous Injections of Fluids into the Trachea, Practitioner, 298, 1886.

Solis-Cohen, S. N. Y. Med. Journ., 1912.

Strübing, P. Deutsche med. Wchnschr., xxxii, 1906.

Strümpell, A. V. Med. Klin., Berlin, July 26, 1908.

Sutherland, G. A. Edinb. Med. Journ., 587, 1906.

——— A System of Diet and Dietetics, 587.

Treupel, G. Deutsche med. Wchnschr., Dec. 31, 1908.

West, Samuel. Lancet, ii, 489, 1908.

——— Clin. Journ., April 1, 1911.

Williams, C. T. Artificial Aero-Therapeutics, C. Allbutt's System of Med., v, 43.

Williams, P. B. Practitioner, Oct., 1910.

Yeo, I. Burney. Food in Health and Disease, 528.

Young, E. S. Hay Fever and Paroxysmal Sneezing, 1910.

——— Hay Fever, Med. Chron., Oct., 1909.

CHAPTER IV

FOREIGN BODIES IN THE BRONCHI

Chevalier Jackson

Foreign bodies in the bronchi have for too long a time been considered curiosities of medicine rather than diagnostic possibilities for exclusion in every acute or chronic case of pulmonary disease. In most of their many phases, foreign body pathology and symptomatology so closely simulate pulmonary disease of other than foreign body origin, that unless the practitioner is alert to the possibilities of foreign body origin, treatment will be unsuccessful, and prognosis grave. Illustrative of this, it may be stated that of 1,170 cases of foreign body at the Bronchoscopic Clinic, the presence of a foreign body as the primary etiologic factor had been overlooked in 102 cases, the foreign body having been present for periods ranging from one month to thirty-six years. In some instances the patient had been in the care of as many as twenty physicians, none of whom suspected a foreign body (Fig. 1).

Pathology of Foreign Bodies in the Bronchi.—The pathologic changes taking place after the lodgment of a foreign body in a bronchus depend chiefly upon two factors: namely, (1) the degree of obstruction to aëration and drainage, and (2) the character of the foreign body. A metallic foreign body, if non-obstructive, will remain in the lungs many months without producing enough pus or secretion to cause a symptom or a râle. A foreign body of this character produces only a strictly local inflammation, of mild degree, and associated with so little suppuration as to suggest a germicidal effect due to electrical or chemical phenomena incident to corrosion. As corrosion proceeds, however, the surface of the foreign body becomes rougher, and hence more and more irritating mechanically by the trauma inflicted. When, after the lapse of months, possibly years, the local accumulation of inflammatory products increases to such an extent that, in combination with the bulk of the foreign body, it obstructs drainage and aëration of the lung tributary to the invaded bronchus, extensive and relatively rapid destructive changes take place. Even in these cases, however, the pathologic processes are of a less aggressive character than those seen in the suppuration following ordinary pulmonary diseases such as influenza and lobar pneumonia. A good illustration of the slow progress in cases of non-obstructive metallic foreign body is shown in Figures 1 and 2.

Glass, pebbles, ivory, hard rubber, and other smooth, hard, insoluble substances, produce very little reaction unless obstructive. In one case at the Bronchoscopic Clinic, a woman had had a glass collar button in her lung since childhood (twenty-six years). Quite a number of years had elapsed before severe lung suppuration became manifest, though when admitted she was almost moribund. The collar button was removed through the mouth, and the patient made a perfect recovery, weighing 182 pounds two years later. We have had many similar cases.

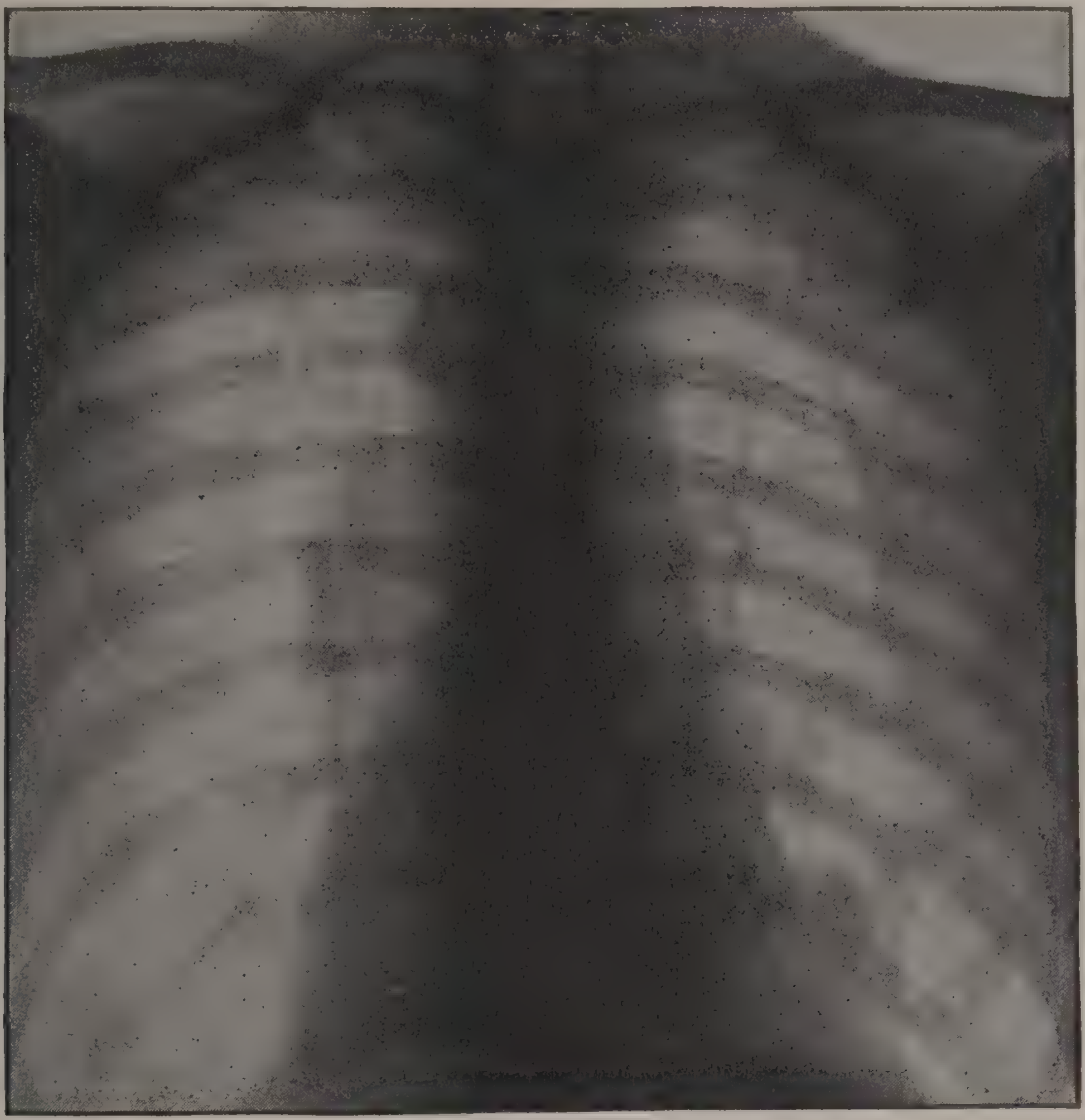


FIG. 1.—CASE NO. FBDY. 726. ROENTGENOGRAM SHOWING SHAWL PIN IN LUNG OF A WOMAN AGED 41 YEARS. This pin produced a complete tuberculous syndrome, including copious hemoptysis. Sojourn of foreign body about 28 years. Pin removed through the mouth in 3 minutes and 25 seconds by bronchoscopy under local anesthesia. Complete recovery followed.

In marked contrast to metallic foreign bodies are vegetable substances, such as nut kernels, maize, apple seeds, fruit-pulp, and seeds of all kinds. It is worthy of note that so septic a thing as a bone, quickly becoming putrid, will, if non-obstructive, give rise to much less local, and no general reaction, while a peanut kernel or a grain of corn will produce in a few days, in children, a violent and diffuse bronchitis.

Arachidic Bronchitis.—This is the name given by the author to a definite disease produced by the aspiration into the bronchi of vegetable substances, especially peanut kernels, of which over 100 cases have come to the Bronchoscopic Clinic. Kernels of maize, heads of grasses, nut kernels, Job's tears, prune and cherry pits, and various other seeds, produce the same reaction and symptoms, but the most frequent offender has been the peanut kernel.

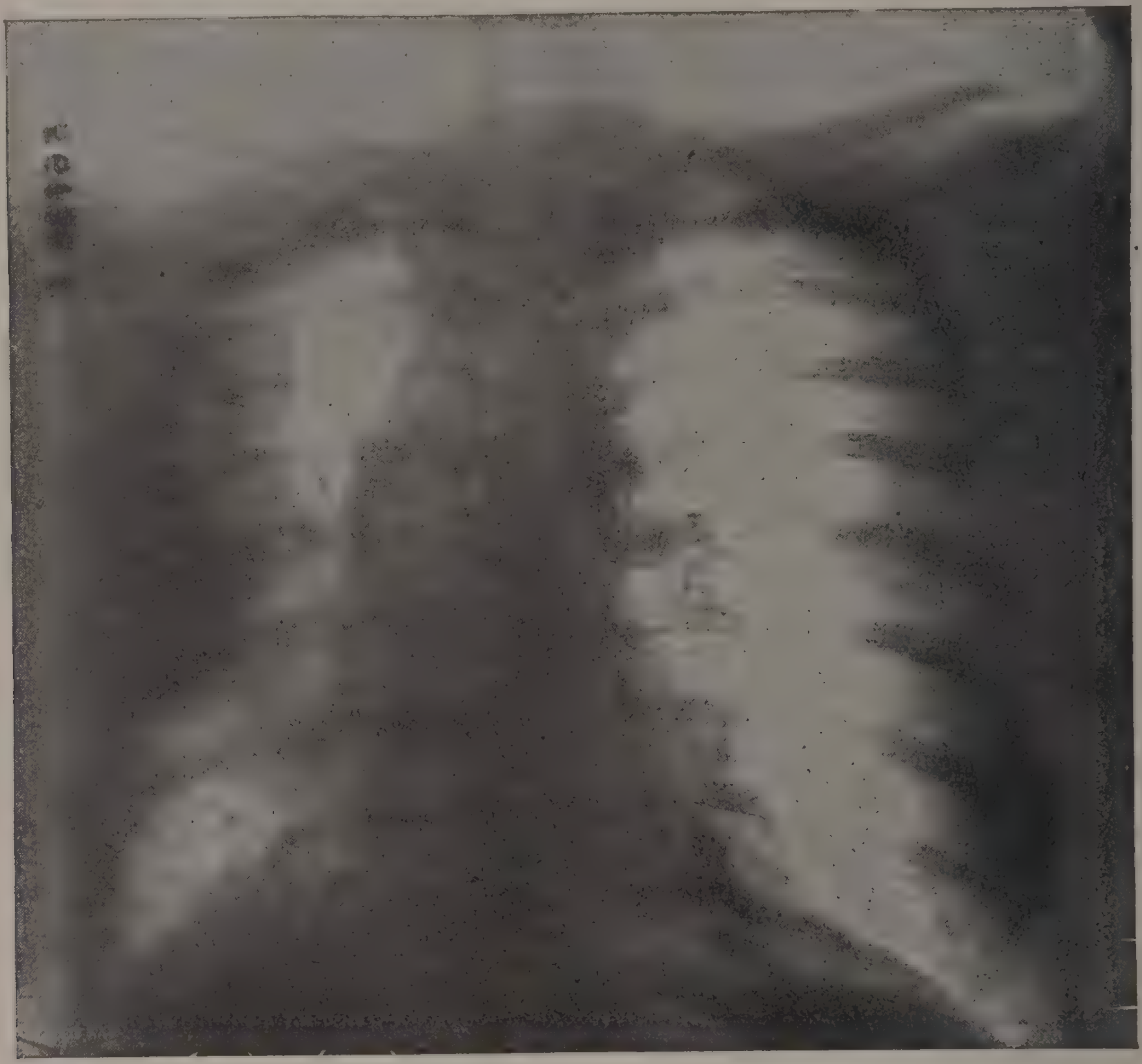


FIG. 2.—CASE NO. FBDY. 1085. ROENTGENOGRAM SHOWING A SAFETY PIN IN THE LUNG OF A WOMAN AGED 46 YEARS. The pin had been in the lung, overlooked, for 36 years. It was removed bronchoscopically through the mouth under local anesthesia in 19 minutes, 7 seconds. Perfect recovery. (From *Medical Clinics of North America*, January, 1923, W. B. Saunders Co.)

The pathologic changes consist of a violent, diffuse, laryngotracheobronchitis, with intense congestion and flooding of the tracheobronchial tree, the violence of the process being in inverse ratio to the age of the child. Adults seem to react but slightly, while an infant will be very ill of toxemia and deficient ventilation of the lung, the obstruction being by both secretion and mucosal swelling. As the disease progresses, the secretions become thicker, and, if not removed by bronchoscopy, plugs of secretion may cause death by mechanical obstruction. Toxemia may be fatal in degree, but more often death is due to occlusion. In children under two years of age, the subglottic edema which always accompanies the tracheobronchitis may asphyxiate the patient if the condition is not realized, and relief afforded. The swelling of the glottic margins prevents proper glottic coöperation in the bechic cycle, in consequence of which the patient may die of autodrownage, that is, he may drown in his own secretions. This has doubtless occurred in many unrecognized cases, in which pneumonia has been thought the cause of death because of the impaired percussion note at the base, due to accumulated secretions. True "lobar pneumonia," as that term is generally used by internists, is seldom if ever a sequel to foreign body in the lung. "Bronchopneumonia" may not be quite so erroneous a term in the peanut cases; the essential difference between the pathologic processes due to foreign body and those due to pulmonary disease of other origin, is the prompt disappearance in the foreign body case, of all the pathology within a few days, in some recent cases within a few hours, after the removal of the intruder. The symptomatology and treatment of arachidic bronchitis are given in subsequent paragraphs.

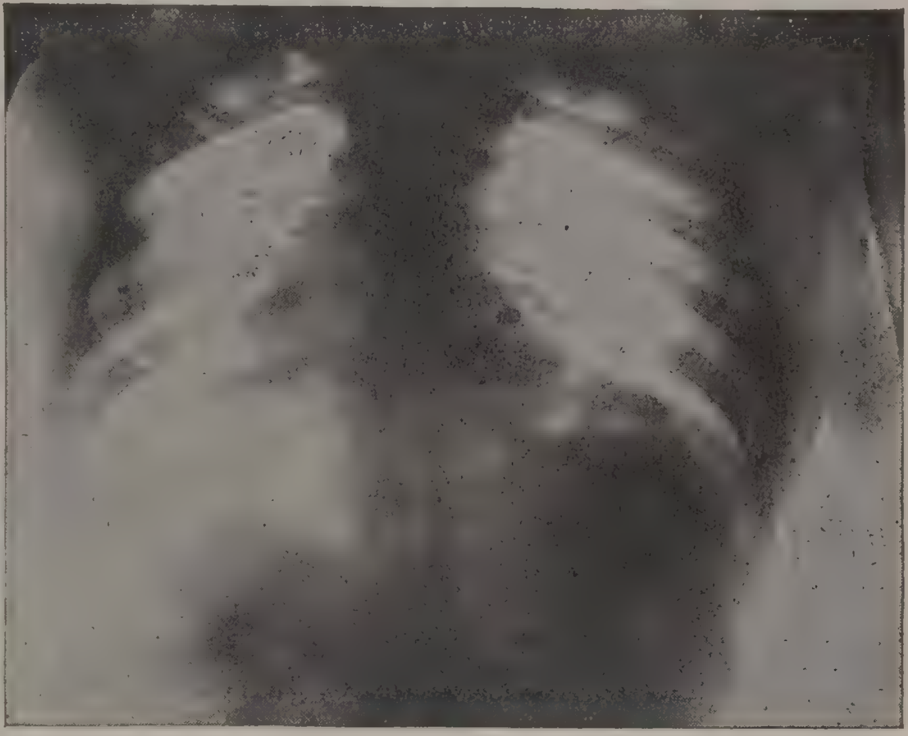


FIG. 3.—CASE NO. FBDY. 266. ROENTGENOGRAM SHOWING BRASS PAPER FASTENER IN THE LUNG OF A WOMAN AGED 23 YEARS. (From *Peroral Endoscopy and Laryngeal Surgery* by Chevalier Jackson.)

Lung-suppuration.—Lung-suppuration due to prolonged sojourn of a foreign body presents certain peculiarities. These are:

1. Foul and dark-colored or greenish pus, indicating the presence of saprophytes and chromogenic bacteria.
2. Presence of an abundant bacterial flora, without distinctly predominant organisms.
3. Absence of bacillus tuberculosis. (Many textbooks have stated that prolonged sojourn of a foreign body ends in tuberculosis.)
4. Presence of a stricture of the bronchus, the foreign body occupying the lumen of the stricture in most cases.
5. The presence of a cavity varying from a slight dilatation of the bronchus to an abscess cavity, though in most instances the "cavity" is filled with granulation tissue, and usually its walls are collapsed. In rare

instances the cavity gapes on inspiration after removal of the foreign body and bronchoscopic aspiration of the pus.

6. The location of the foreign body is usually at the upper part of the pathologic tissue which has resulted from its presence.

When the drainage from a bronchus, large or small, becomes obstructed, the secretions accumulate below, and we find the natural passages filled with secretions which are, or soon become, purulent. This condition we have fallen into the custom of referring to as "drowned lung." It may give the same note as an atelectatic area, and the two conditions may coexist. Clinically, it differs from true abscess in that it may develop rapidly, within a day or two in some instances, and in that, if relieved at once by removal of the foreign body, recovery from whatever local reaction may be present is almost always prompt and complete. If, however, the area of "drowned lung" is not relieved by the removal of the foreign body from the bronchus, the bacteria become active in the pent-up secretions; the saprophytes decompose the secretions into irritant products, the toxins and other products of bacterial activity, together with the inflammation, result in destruction of the cilia, the epithelium, then the subepithelial tissue, and the perichondrium; the cartilage of the rings dies away, leaving a cavity. Across such a cavity may be stretched bands of tissue, often branched; these may be bronchi, vessels or fibrous tissue.

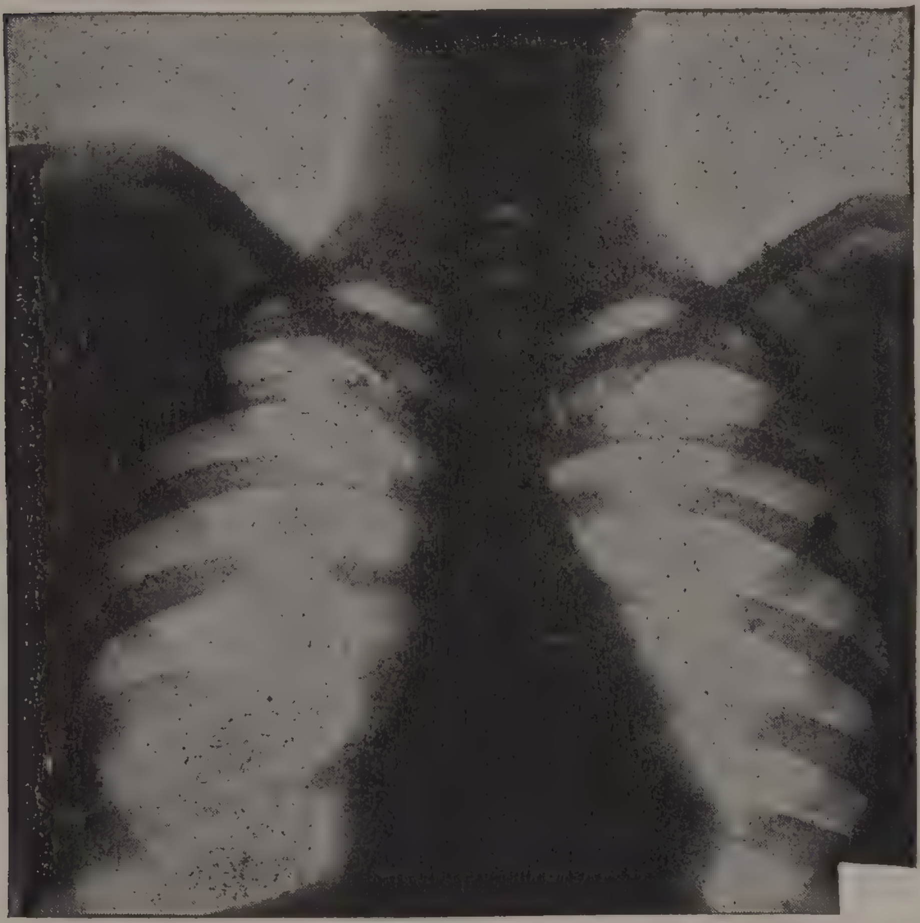


Fig. 4.—Same case as in Fig. 3. Seven Years after Peroral Bronchoscopic Removal of Brass Paper Fastener That Had Been in the Lung Seven Years. There are no more pathologic shadows than are usual in the average individual of the same age. No expectoration or other pulmonary symptom remained after the few months of convalescence required for the suppuration to cease.

One very striking feature of pulmonary abscess due to foreign body is that practically all cases recover promptly after removal of the foreign body, with no treatment other than an outdoor rest régime. This strong tendency to recover, as compared with cases of postpneumonic and postinfluenzal abscess points to a different pathologic process. At the Bronchoscopic Clinic, out of the 116 traceable cases of lung-suppuration due to the sojourn of a foreign body for periods of from one month to thirty-six years, 110 are alive and, of the 110, all of those bronchoscoped prior to one year ago are in good health. These are long sojourn cases. In cases of recently aspirated foreign bodies there have been recoveries in 98.3 per cent.

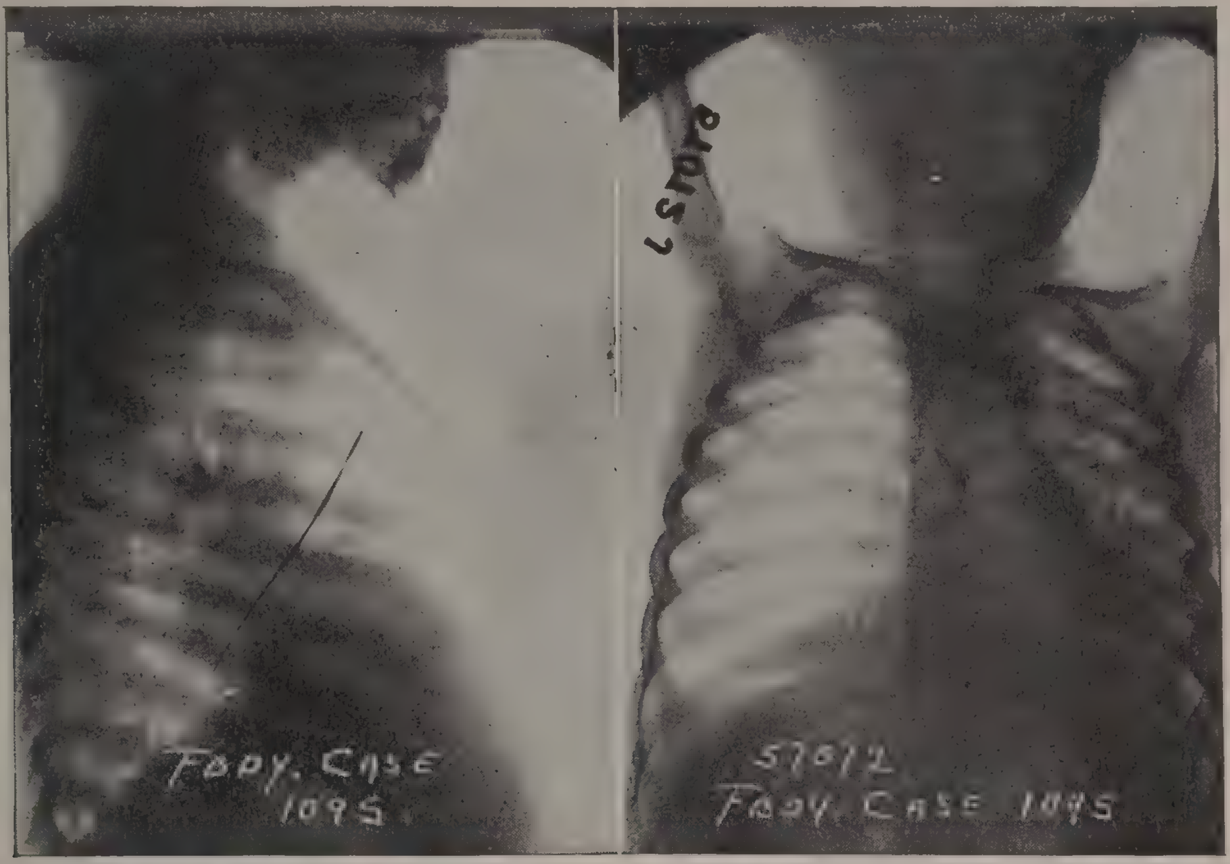


Fig. 5.—Case No. Fbdy. 1095. Roentgenograms, Anteroposterior and Lateral, of a Child Aged 23 Months, Showing Pulmonary Abscess Caused by the Unknown Presence of a Screw for 20 Months. Removed bloodlessly by bronchoscopy through the mouth, without anesthesia general or local, in 3 minutes and 16 seconds. (Films by Dr. Willis F. Manges.) (From *Medical Clinics of North America*, January, 1923, W. B. Saunders Co.)

SYMPTOMATOLOGY, DIAGNOSIS, AND TREATMENT OF FOREIGN BODY IN THE AIR PASSAGES

Symptomatology

From an experience of over 1,000 cases of foreign body in the air and food passages, the author has worked out a plan of procedure to be pursued in every case of suspected foreign body; and foreign body should be suspected in every case of acute or chronic disease of the larynx, trachea, or lungs. This plan has been taught for many years to the physicians and surgeons who have taken the author's courses in bronchoscopy and esophagoscopy, and has been modified by the collective experience of all the pupil-physicians who have taken these courses. The following is the résumé of the symptomatology and diagnosis of foreign body in the air and food passages that is given to every pupil-physician coming for instruction to the Bronchoscopic Clinic. Laryngeal and esophageal diagnosis are both included, since the diagnosis is often a matter of differentiation between esophageal and tracheobronchial lodgment of a foreign body, both laryngeal and esophageal lodgment often producing bronchial symptoms.

Initial Symptoms.—The most important initial symptoms are choking, gagging, coughing and wheezing, often followed by a symptomless interval. A foreign body may be in the larynx, trachea, bronchi, nasal chambers, nasopharynx, fauces, tonsil, pharynx, hypopharynx, esophagus, stomach, intestinal canal, or may have been passed by bowel, coughed out or spat out, with or without knowledge of the patient. Initial choking, etc., may have escaped notice or may have been forgotten.

Laryngeal Foreign Body.—One or more of the following laryngeal symptoms may be present: hoarseness, croupy cough, aphonia, odynphagia, hemoptysis, wheezing, dyspnea, cyanosis, apnea, subjective sensation of foreign body. Croupiness usually means subglottic swelling. Obstructive foreign body may be quickly fatal by laryngeal impaction on aspiration, or on abortive bechic expulsion. Lodgment of a non-obstructive foreign body may be followed by a symptomless interval. Direct laryngoscopy for diagnosis is indicated in every child having laryngeal diphtheria without faucial membrane. (No anesthetic, general or local, is required.) In the presence of laryngeal symptoms we should, I think, consider the following possibilities:

1. A foreign body in the larynx.
2. A foreign body loose or fixed in the trachea.
3. Digital efforts at removal.
4. Instrumentation.
5. Overflow of food into the larynx from esophageal obstruction due to foreign body.
6. Esophagotracheal fistula from ulceration set up by a foreign body in the esophagus, followed by leakage of food into the air-passages.
7. Laryngeal symptoms may persist from the trauma of a foreign body that has passed on into the deeper air or food passages or that has been coughed or spat out.
8. Laryngeal symptoms (hoarseness, croupiness, etc.) may be due to digital or instrumental efforts at removal of a foreign body that never was present.
9. Laryngeal symptoms may be due to acute or chronic laryngitis, diphtheria, pertussis, infective laryngotracheitis and many other diseases.
10. Deductive decisions are dangerous.
11. If the Roentgen ray is negative, laryngoscopy (direct in children,

indirect in adults) without anesthesia, general or local, is the only way to make a laryngeal diagnosis.

12. Before doing a diagnostic laryngoscopy, preparations should be made for taking a swab-specimen and for bronchoscopy and esophagoscopy.

Tracheal Foreign Body.—(1) "Audible slap," (2) "palpatory thud," and (3) "asthmatoid wheeze" are pathognomonic. The "tracheal flutter" has been observed by McCrae. Cough, hoarseness, dyspnea and cyanosis are often present. Diagnosis is by Roentgen ray, auscultation, palpation, bronchoscopy. The "asthmatoid wheeze" is heard with the ear or stethoscope bell (McCrae) at the patient's open mouth, not at the chest wall. Listen long for the "audible slap," also heard at the open mouth. It is the name given by the author to the peculiar sound caused by the sudden arrest of the foreign body by the subglottic narrowing, during nature's expiratory or bechic attempt at expulsion of the intruder. When felt

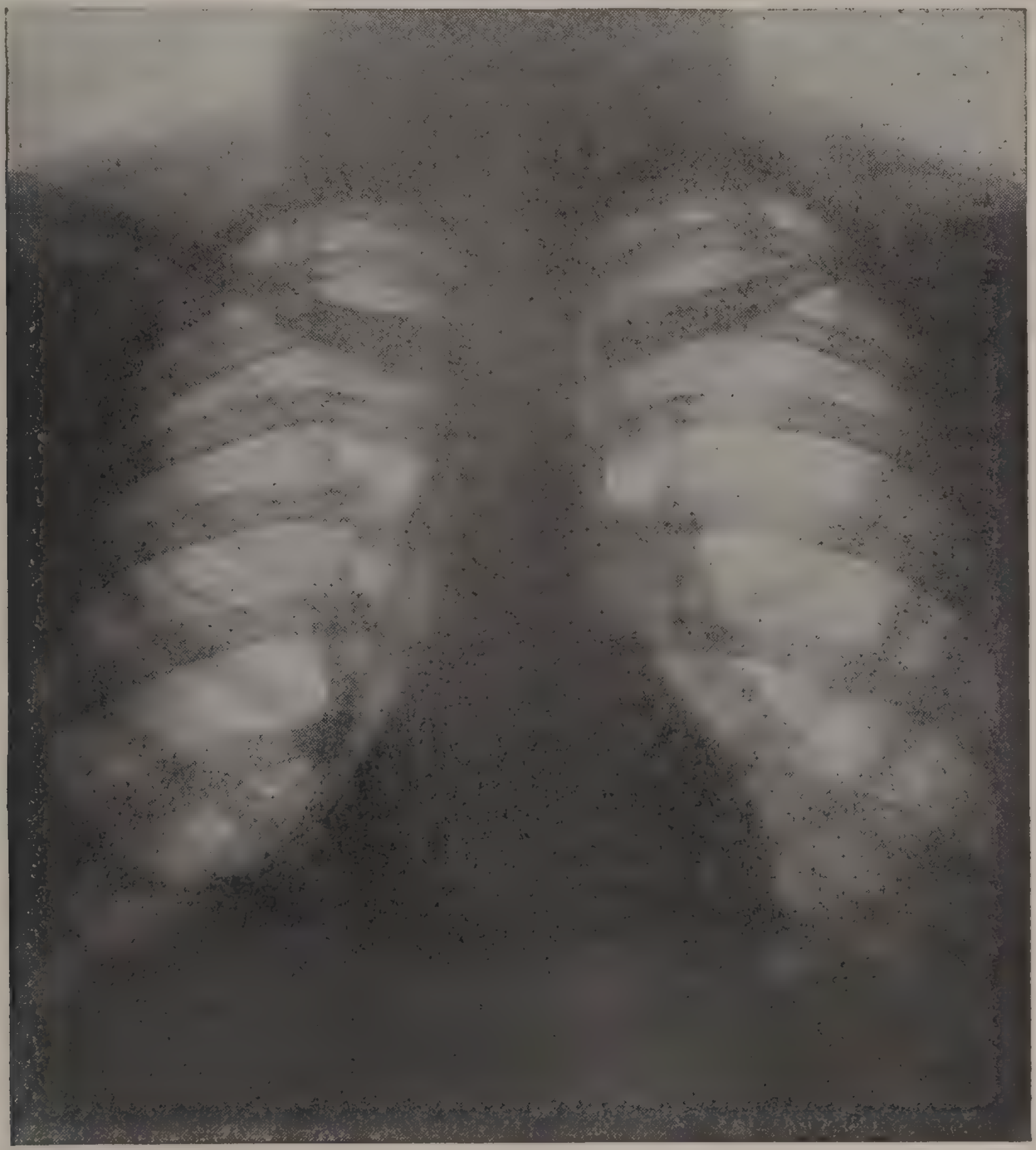


Fig. 6.—A Case of Total Absence of the Cough Reflex Despite the Presence, During a Period of 2 Months, of a Safety Pin in the Lung of a Woman Aged 18 Years. The inference is that afferent bechic impulses are not generated in this peripheral portion of the lung after tolerance is established. Productive cough started 2 months after aspiration of the pin. (Roentgenogram by Dr. James F. Kelly.)

by the examiner's thumb on the trachea it constitutes the sign that the author has called the "palpatory thud." History of initial choking, gagging, and wheezing is important if elicited, but is valueless negatively.

Bronchial Foreign Body.—Initial symptoms are coughing, choking, asthmatoid wheeze, etc., noted above. There may be a history of these or of tooth extraction. At once, or after symptomless interval (Fig. 6), cough, blood-streaked sputum, metallic taste, or special odor of foreign body may be noted. Non-obstructive metallic foreign bodies afford few symptoms and few signs for weeks or months (Fig. 7). Obstructive foreign bodies cause atelectasis, drowned lung and eventually pulmonary abscess. Lobar pneumonia is an exceedingly rare sequel. Vegetable organic foreign bodies such as peanut kernels, beans, watermelon seeds, and the like cause at once violent laryngotracheobronchitis (arachidic bronchitis) with toxemia, cough and irregular fever, the gravity and severity being inversely to the age of the child. Bones and metallic bodies after months or years produce changes which cause chills, fever, sweats, emaciation, clubbed fingers, incurved nails, cough, foul expectoration, hemoptysis; in fact all the symptoms of chronic pulmonary sepsis, abscess or bronchiectasis, and many signs which may suggest pulmonary tuberculosis. The apices, however, are normal and bacilli are absent from the sputum.

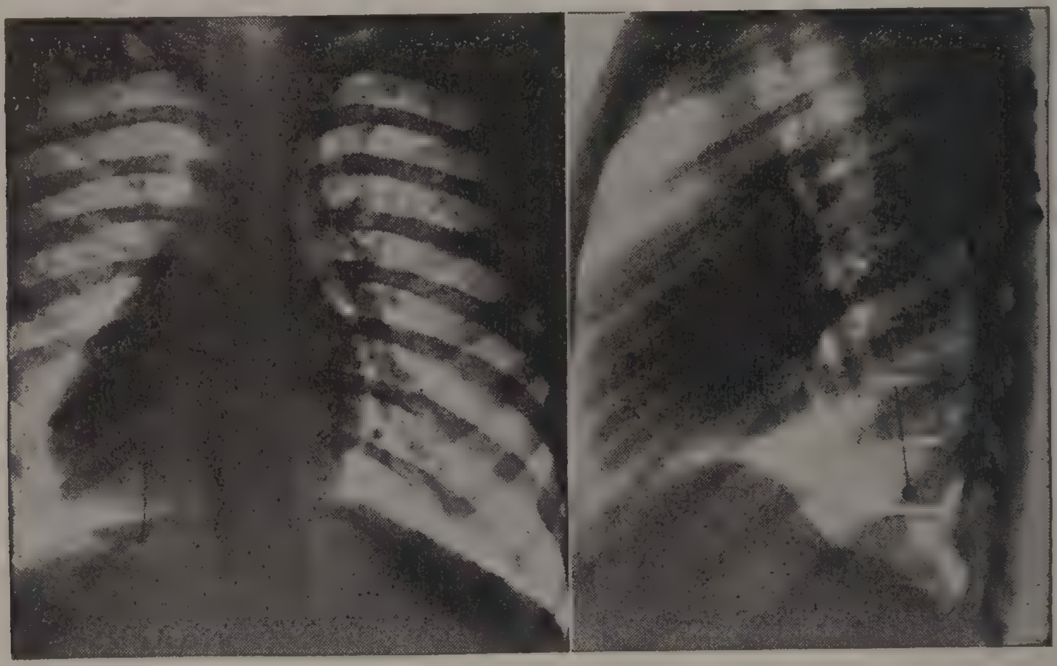


Fig. 7.—A Case of Absence of Cough Despite the Presence of a Shawl Pin Deep in the Left Lower Lobe (Patient a Girl Aged 10 Years). The apparently warranted inference is that the periphery of the lung is not a source of afferent tussive impulses. This seems curious in view of the fact, observed in this and other cases by Professor Thomas McCrae, that unilateral limitation of expansion without bronchial obstruction indicates the presence of afferent non-tussive impulses.

It must always be borne in mind that a foreign body lodged in the esophagus may present many of the signs of bronchial foreign body. This is caused in two ways:

(*a*) A foreign body in the esophagus by the obstruction to swallowing may cause foods, liquids and saliva to overflow into the larynx whence they are aspirated into the lungs.

(*b*) A foreign body lodged in the esophagus may in time ulcerate its way into the trachea producing a fistula through which food, liquids and saliva leak directly into the lower air passages. The symptoms of pulmonary disease are violent in either form, and if the foreign body is not removed, death quickly follows.

DIAGNOSIS

The Roentgen Ray.—The Roentgen ray is the most valuable diagnostic means; but careful notation of physical signs by an expert is, in all cases, essential. Expert ray work will show all metallic foreign bodies and many of less density, such as teeth, bones, shells, buttons, etc. If the ray is negative, a diagnostic bronchoscopy should be done in all cases of unexplained bronchial obstruction.

Peanut kernels, maize, watermelon seeds, and other vegetable substances in the bronchi produce obstructive emphysema of the invaded side. Fluoroscopy shows the diaphragm flattened, depressed, and of less excursion on the invaded side; at the end of expiration, the heart and mediastinal wall move over toward the uninvaded side, and the invaded lung becomes less dense than the uninvaded lung, from trapping of the air by the expiratory, valvelike effect of obliteration of the "forceps spaces" that during inspiration afford air ingress between the foreign body and the swollen bronchial wall. This partial obstruction causes *obstructive* emphysema, which must be distinguished from *compensatory* emphysema, in which the ballooning is in the unobstructed lung, because its fellow is wholly out of function through *complete* "corking" of the main bronchus of the invaded side (Figs. 8 and 9). The phenomenon of obstructive emphysema, first discovered by Iglauer, has been developed by Manges to such a high degree of perfection that he has been able in over 100 cases to tell me with absolute accuracy that a peanut kernel or a grain of maize or other nonopaque vegetable substance was present in the lung at a certain definite location with reference to the bronchial tree. Also, in a number of cases of incomplete removal, he has been able to tell me that a fragment remained behind and on this information I have done a second bronchoscopy and found the remaining fragment in the location specified. The fluoroscope is not used during the bronchoscopy.

Physical Signs.—Secretions, normal and pathologic, may shift from one location to another; the foreign body may change position admitting more, less, or no air, or it may shift to a new location in the same lung or even in the other lung. A recently aspirated pin may produce no signs at all. The signs of diagnostic importance are chiefly those of partial

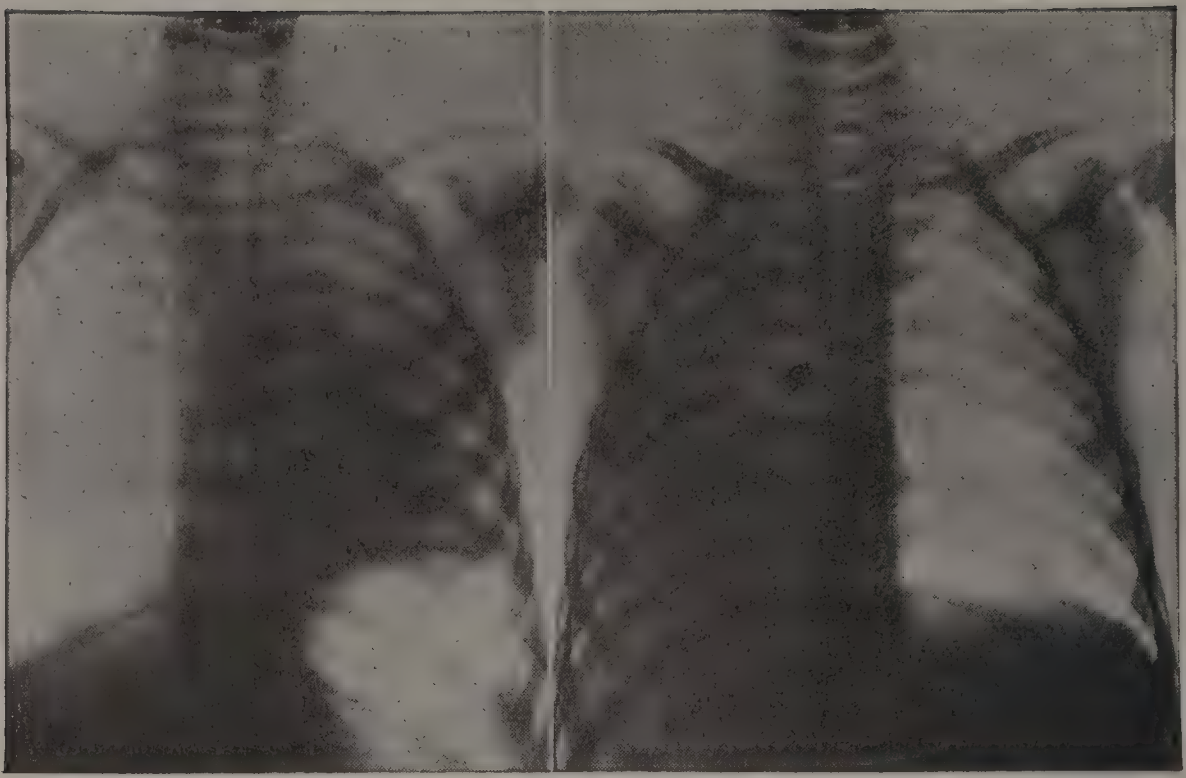


Fig. 8.—Case No. Fbdy. 645. Roentgenogram Showing in the Right Hand Illustration a Small Electric Lamp in Left Lung of a Boy Aged 6 Years. Left main bronchus tightly "corked." Lamp in situ six days; left lung atelectatic; right lung emphysematous (compensatory emphysema). Mediastinum displaced to atelectatic side. These changes were demonstrable by physical signs. Lamp was removed without anesthesia in 1 minute 9 seconds. Roentgenogram at the left was taken 10 minutes after removal, and shows air beginning to enter the collapsed lung. Recovery complete in 24 hours. Symptoms: fever, cough, dyspnea, distress. At first thought to be pneumonia. Physical signs: Greatly limited expansion on the left side. Impaired resonance above and flatness below the fourth rib, front and back. Breath sounds intensely tubular, with few fine crackling râles above fourth rib. Below breath sounds very distant and vocal resonance much diminished. Right side of chest hyperresonant, with harsh breath sounds and numerous loud harsh râles. (Plates by Arthur C. Sender.)

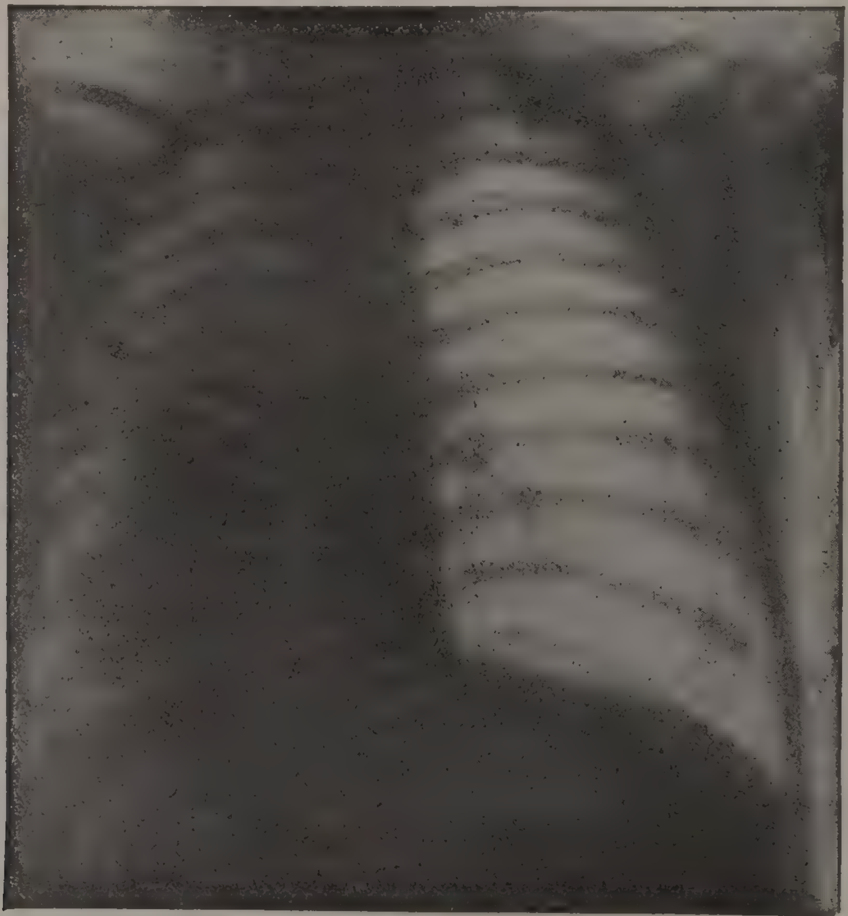


Fig. 9.—Expiratory Valvelike Bronchial Obstruction by Non-radiopaque Foreign Body, Producing an Acute Obstructive Emphysema. Peanut kernel in right main bronchus. Note (*a*) depression of right diaphragm; (*b*) displacement of heart and mediastinum to left; (*c*) greater transparency of the invaded side. (Roentgenogram by Dr. Willis F. Manges.) (From *Bronchoscopy and Esophagoscopy Textbook*, W. B. Saunders Co.)

or complete bronchial obstruction, though a non-obstructive foreign body, a pin, for instance, may cause limited expansion (McCrae) or, rarely, a peculiar râle or a peculiar auscultatory sound. The most nearly characteristic physical signs are:

1. Limited expansion.
2. Decreased vocal fremitus.
3. Impaired percussion note.
4. Diminished intensity of breath-sounds distal to the foreign body.

Complete obstruction of a bronchus followed by drowned lung adds absence of vocal resonance and vocal fremitus, thus often leading to an erroneous diagnosis of empyema. Varying grades of tympany are obtained over areas of obstructive or compensatory emphysema. With complete obstruction there may be tympany from collapsed lung for a time. Râles in case of complete obstruction are usually most intense on the uninvaded side. In partial obstruction they are most often found on the invaded side distal to the foreign body, especially posteriorly, and are often most intense at the site corresponding to that of the foreign body. A foreign body at the bifurcation of the trachea may give signs in both lungs. Early in a foreign body case, diminished expansion of one side, with dulness, may suggest pneumonia in the affected side; but the decreased vocal fremitus and the diminished breath-sounds with absence of or decreased vocal resonance, and absence of typical tubular breathing should soon exclude this diagnosis. Bronchial obstruction in pneumonia is exceedingly rare. As stated by McCrae:

"There is no one description of physical signs which covers all cases. If the student will remember that complete obstruction of a bronchus leads to a shutting off of this area, there should be little difficulty in understanding the signs present. The diagnosis of empyema may be made, but the outline of the area of dulness, the fact that there is no shifting dulness and the greater resistance which is present in empyema nearly always clear up any difficulty promptly. The absence of the frequent change in the voice sounds, so significant in an early small empyema, is of value. A large empyema should give no difficulty. If difficulty remains, the use of the needle should be sufficient. In thickened pleura, vocal fremitus is not entirely absent, and the breath-sounds can usually be heard, even if diminished. In case of partial obstruction of a bronchus, it is evident that air will still be present, hence the dulness may be only slight. The presence of air and secretion will probably result in the breath-sounds being somewhat harsh, and will cause a great variety of râles, principally coarse, and many of them bubbling. Difficulty may be caused by signs in the other lung or in a lobe other than the one affected by the foreign body. If it is remembered that these signs are likely to be only on auscultation, and to consist largely in the presence of râles, while the signs in the area supplied by the affected bronchus will include those on inspection, palpation and percussion, there should be little difficulty."

Esophageal Foreign Body.[1]—After initial choking and gagging, or without these, there may be a subjective sense of a foreign body, constant or, more often, on swallowing. Odynphagia and dysphagia or aphagia may or may not be present. Hematemesis and fever may occur from the foreign body or from rough instrumentation. Symptoms referable to the air-passages may be present due to: (1) overflow of secretions on attempts to swallow through the obstructed esophagus; (2) erosion of the foreign body through from the esophagus into the trachea; (3) compression of the trachea by a large foreign body in the esophagus; or (4) trauma inflicted on the larynx during attempts at removal, digital or instrumental, the foreign body still being present or not.

Diagnosis is by the Roentgen ray, first without, then, if necessary, with a capsule filled with an opaque mixture. Flat objects, like coins, always lie with their greatest diameter in the coronal plane of the body, when in the esophagus; in the sagittal plane when in the trachea. Lateral, anteroposterior, and sometimes also quartering roentgenograms are necessary. One taken laterally, low down on the neck but clear of the shoulder, will often show a bone not visible in the anteroposterior exposure.

The foregoing outline will afford the reader a general grasp of the subject. We will now consider some details at greater length.

The following separate consideration of each class of physical signs of foreign body in the lung by McCrae is based upon a relatively large experience at the Bronchoscopic Clinic.

Inspection.—In every case seen by me there has been definite diminished expansion on the affected side. If the foreign body has shifted from one side to the other, or is situated at the bifurcation of the trachea, the expansion may be decreased on both sides. In some cases in which the foreign body was a pin, decreased expansion was the only sign. If the foreign body has acted as a valve and allowed air to enter but not to escape, the affected part of the lung will be distended and that part of the thorax be fuller—but the expansion is less. The extent of movement of the diaphragm is important to note.

Palpation.—The vocal fremitus varies with the condition present. If a bronchus is completely plugged, vibrations will be absent over the supplied portion of lung. If the closure is intermittent the vibrations may be absent at one time and present at another. If the bronchus is partially obstructed, there may be a decrease in the vibrations. In young children it may not be possible to gain much information from the study of the fremitus.

---

[1] Though esophagoscopy is not within the scope of this article, this portion of the summary is included because of the necessity of exclusion of esophageal lodgment in any case suspected of foreign body in the bronchi.

Percussion.—Evidently the findings must vary greatly. With a valve-like action of the foreign body the affected portion of lung becomes markedly emphysematous and hence yields hyperresonance or tympany. With complete plugging of a bronchus there will be flatness over the affected portion of lung as soon as all the contained air is absorbed. If the plugging is not constant there may be some resonance at one time and none at another, but there is usually some grade of dulness. The sense of resistance will vary with the condition present. With collapse of a portion of lung there is likely to be tympany for a time. There may be varying grades or combinations of dulness and tympany, especially in children, in whom hyperresonance and tympany are common. These are often very confusing, but a careful comparison with the note elsewhere will usually lessen the difficulty.

Auscultation.—The most diverse findings are to be expected and the signs may vary from hour to hour if the bronchus is not completely plugged. The degree of collapse of the lung, the amount of contained air and fluid, the extent of fibroid change, the presence of abscess or bronchiectasis, all influence the signs. Over a lobe the bronchus of which is completely plugged, as a rule the breath sounds are absent but occasionally, and especially in children, distant breath-sounds may be heard. Over the portion of lung supplied by a partially obstructed bronchus, the breath-sounds are harsh and rough with prolonged expiration, accompanied by many râles, usually coarse, sometimes bubbling and with both inspiration and expiration. Over other parts of the lung of the affected side, and on the other side, the findings depend on the amount of irritation set up and the presence of secretion. Râles may be heard everywhere in the acute cases. In some cases of a small metallic foreign body in a bronchus, not sufficiently large to cause any marked obstruction, very fine râles described by older authors as "tissue paper" râles may be heard. They are heard at the end of inspiration, and are much finer than the fine crepitations heard at the early stage of lobar pneumonia.

TREATMENT

Coughing Up Foreign Bodies.—Waiting for the foreign body to be coughed up is not justfiable, because this happy result occurs in only about 2 per cent of foreign bodies that have reached the bronchi. In the pre-bronchoscopic days, the formidable operation of thoracotomy, with its high mortality, fully justified waiting. But to-day, when 98 per cent of foreign bodies can be quickly and safely removed through the mouth without any anesthetic, general or local, waiting for bechic expulsion is not seriously to be considered.

Disintegration of Foreign Bodies.—Waiting for a foreign body to disintegrate is hopeless except in cases of pure candy or similar soluble substance. This does not include nut candies. Peanut kernels, grains of maize, etc., do not disintegrate by maceration. Steel objects corrode and rust, but do not disappear, as shown by the case illustrated in Figure 1, in which a pin made of highly tempered steel, the most easily corroded of metals, still remained as a foreign body.

Thoracotomy for Foreign Body.—Since bronchoscopy has developed to such a point that any localizable foreign body that has gone down the natural passages can be removed by the same route, thoracotomy is no longer justifiable. Its high mortality renders it much more dangerous than letting the foreign body alone indefinitely. It no longer has a place even in cases remote from a bronchoscopist, because there is ordinarily no urgency about removing a foreign body unless asphyxia is threatened. In such a case tracheotomy could be done to prevent asphyxia until a bronchoscopist could be reached. It was thought by some surgeons that pulmonary abscess, due to prolonged sojourn of a foreign body, would require external drainage, but abundant clinical experience has shown that practically all cases of lung-suppuration of even the longest standing clear up completely after bronchoscopic removal of the foreign body. At the Bronchoscopic Clinic, as cited in a preceding paragraph, nearly all of 116 cases of pulmonary suppuration (abscess and bronchiectasis) cleared up completely after bronchoscopic removal of the foreign body, dilatation, and bronchoscopic removal of the granulations and pus (Figs. 1, 2, 3, 4 and 5).

Treatment after Bechic Expulsion of a Bronchial Foreign Body.—After a foreign body has been coughed up, if it has been recently aspirated, ordinarily no treatment is required. Antibechics, such as codein, heroin, morphin, etc., are particularly pernicious, as they check the cough reflex, which is the watch-dog of the lungs, and will, if not drugged, quickly rid the lung of any infective agents or inflammatory products. If the foreign body has been in the lung for a long period, the only treatment ordinarily required is a fresh air régime, that is, rest in bed outdoors for from twelve to fourteen or more hours a day, according to the condition of the patient. If the physical signs and fever show obstructed drainage of any area of lung, bronchoscopic removal of granulations, bronchoscopic dilatation of a strictured bronchus, or bronchoscopic aspiration of the purulent accumulations, may be necessary, but are usually not required.

Bronchoscopic Removal of Foreign Bodies from the Lungs.—The exceptions are so rare that it may be stated as an axiom that any localizable foreign body of appreciable size that has gone down the natural passages can be brought back the same way, though perhaps not at the first attempt. If mortality is avoided, however, patient, concentrated endeavor will solve any problem of bronchoscopic foreign body removal. The only exception to this broad and sweeping assertion as to the unlimited possibilities of bronchoscopic foreign body extraction is in the case of foreign bodies that are so small as not to be localizable. It is obvious that a minute piece of pneumoconiotic material, or a tiny fragment of peanut kernel, for instance, may get into a tiny branch bronchus and therefore cannot be located. It is impossible thoroughly to search the hundreds of minute bronchi and bronchioles of both lungs for tiny particles of foreign bodies that are non-opaque and hence not localizable. Non-opaque bodies of appreciable size can be located by the ray, and by the physical signs.

Bronchoscopy is a procedure using a straight and rigid tube that, when inserted into the trachea and bronchi by way of the mouth, serves as a speculum. An aspirating tube in the wall of the bronchoscope removes pus and secretions, and a tiny lamp at the distal end of the instrument furnishes illumination. Through the tube, long slender forceps and other instruments can be inserted for the removal of foreign bodies and the treatment of disease. The introduction of the bronchoscope does not require general anesthesia. In adults, a little cocain may be applied to the mucosa of the laryngopharynx, using a 10 per cent solution, and also to the interior of the trachea. In children, however, cocain is dangerous for any purpose, and quite unnecessary. A little morphin hypodermically or paregoric internally may be given to lessen cough if desired. General anesthesia is not necessary, but is not contra-indicated except in dyspneic cases, in which it introduces a large element of danger.

For the safe and successful performance of bronchoscopy, an adequate instrumentarium, special training, and trained assistants are necessary. For a description of the technic, the reader is referred to special works on the subject.

As there is now in practically every city one or more men trained and equipped for the work, and as, moreover, the cases are rarely too urgent or in too bad condition to be sent to the bronchoscopist, the general practitioner is not obliged to be prepared to do bronchoscopy. An exception, however, should be made in cases of extreme dyspnea; in these cases low tracheotomy should be done, not for the insertion of a bronchoscope, but for the relief of asphyxia. It must be remembered that a foreign body may be dislodged by cough from a bronchus, and be impacted in the subglottic region, an accident likely to happen as a result of the discredited procedure of holding the patient up by the ankles.

After the bronchoscopic removal of a foreign body, in recent cases, the after-care should consist of rest in bed for a few days, hospitalization being as a rule unnecessary. In cases of prolonged sojourn of a foreign body in the lung, with extensive suppuration, abscess, etc., after removal of the intruder the patient should have rest in bed outdoors sixteen or more hours out of the twenty-four. The patient should not waste his energies, but build up a reserve strength for resistance. The food should be plain, nutritious, and ample. Elimination of toxins should be promoted by careful attention to the bowels, skin, and kidneys. There may be an occasional case in which a bronchial stricture from cicatricial contraction may require bronchoscopic dilatation, but such a sequel is very rare.

Bronchoscopic Removal of Penetrating Projectiles.—Bronchoscopic removal of the penetrating projectiles from the lungs is quite feasible in cases where the projectile is not too large to be brought out through the corresponding main bronchus without rupturing it. Long foreign bodies, if the lesser diameter is not too large, may be turned and brought out endwise.

REFERENCES

Jackson, Chevalier. Mütter Lecture. Pathology of Foreign Bodies in the Air and Food Passages, Surg., Gynec. & Obst., March, 1919.

——— Peroral Endoscopy and Laryngeal Surgery, 1914. Clinics of North America, Jan., 1923. Bronchoscopy and Esophagoscopy, Textbook, W. B. Saunders Co., 1923.

Manges, Wm. F. Diagnosis and Localization of Non-opaque Foreign Bodies in the Bronchi, Am. Journ. Roent., No. 6, vii, 1920.

McCrae, Thomas. The Diagnosis of Foreign Bodies in the Bronchi, Am. Journ. Med. Sc., March, 1920.

McCrae, Thomas. The Clinical Features of Foreign Bodies in the Bronchi. Lumleian Lectures, Lancet, April 12, 19 and 26, 1924.

CHAPTER V

DISEASES OF THE LUNGS

Philip H. Pierson and George Blumer

CIRCULATORY DISTURBANCES

Philip H. Pierson

To understand properly circulatory disturbances in the lungs it is necessary to have clearly in mind a few of the essential factors which influence the flow of blood through the lungs. The right and left ventricle must exactly keep pace with each other, putting out each the same amount of blood in the same given time, except for very occasional short intervals, or pathology will so quickly develop that death will ensue. In spite of the fact that the same amount of blood is put into both the systemic and pulmonary circulations with each cardiac beat, the pressure in the pulmonary arteries averages only 18 to 20 mm. Hg., frequently varying 12 mm. from inspiration to expiration, at times rising as high as 48 mm. Hg., while the average systemic systolic arterial pressure averages 110 to 120 mm. Hg. Pulmonary diastolic pressure averages 12 mm. Hg., and varies about 6 mm. with respiration. This considerable difference is due to the large caliber of the pulmonary vessels, their close proximity to the heart, requiring no great load of work to be done and their relative straight direction. With each inspiration the normal elasticity of the lung creates a suction within the vena cava amounting to 4 to 7 mm. Hg., which facilitates the return flow of the blood to the heart. With expiration this suction action is retarded considerably but does not wholly disappear. Deep and rapid breathing also creates an increased flow of blood from the abdominal veins through the right heart into the pulmonary system. The elastic recoil of the great vessels upon their valves is another factor which aids the constancy of the pulmonary circulation. It is probable that there is some vasomotor control of the blood-vessels in the lungs but only rarely in health does this influence the size of the vessels. Under the subject of pulmonary edema, this will be further discussed. The strength of the left ventricle and its ability to expel blood into the general circulation is likewise a factor in the normal flow through the lungs.

Certain factors interfere with this normal pulmonary circulation and these are abnormal consistency of the blood, infections, stenosis or prolonged and decompensated insufficiency of the mitral or aortic valves and insufficiency of the tricuspid valves, weakness of the heart muscle, abnormally high systemic pressure and pressure by some mass on large branches of the pulmonary arteries or veins. Such congenital anomalies as patent foramen ovale or ductus arteriosus naturally change the normal flow of venous and arterial blood in the lungs, favoring poorer aëration of the blood and its consequent effect on the lungs and general system. Drugs also influence the pulmonary circulation but chiefly by their action on the heart or the peripheral circulation.

Increased venous pressure occurs when the systemic pressure is excessively high and the left ventricle weakens. This in turn causes more resistance in the capillaries and a rise in arterial pressure follows. Mechanically, the circulation in health is so adjusted that variations in the heart rate seldom cause congestion or depletion of the circulation in the lungs.

Pulmonary pressure is also increased by rapid intravenous saline injection during which the right heart puts more blood into the lungs than the left can pump out. Dyspnea increases pulmonary pressure by drawing an increased amount of blood from the abdominal veins and forcing it through the thin-walled right ventricle. Stenosis or insufficiency of the mitral or aortic valves increases pulmonary pressure which is maintained until either the left heart is restored to its efficiency or the right heart weakens and the congestion is distributed to the systemic circulation causing passive congestion in the liver, kidneys, spleen and peripheral vessels.

Lowering of pulmonary pressure may be accomplished by hemorrhage either from the lungs or some peripheral vessel, by constriction of the extremities, thereby diminishing the amount of blood returnable to the heart, by dilatation of the peripheral vessels, and by drugs acting on the heart or systemic system. Pituitrin, when injected hypodermically, causes a rise in systemic systolic and diastolic pressure and a consequent rise in pulmonary pressure, especially the systolic. Pulmonary pressure rises with the administration of hydrastin because of the fall in systemic pressure and the weakness of the left heart, thus damming back blood in the pulmonary circuit and causing engorgement. Wiggers, working with ergotoxin, found that the pulmonary arterial pressure was first reduced and then raised, while the pressure in and the hemorrhage from the veins was diminished, owing to a constriction of the smaller pulmonary vessels. After severe hemorrhage both these effects pass off. Epinephrin, when producing definite effects upon the pulmonary pressure, has raised both the arterial and venous pressure, and has increased hemorrhages from the arteries, veins and capillaries. A fall of pressure in the left auricle is attributed by Wiggers to the increased activity of the left heart. According to most observers, digitalis and related drugs produce similar, though less marked effects.

Nitroglycerin and the nitrites, by their dilating effect on the peripheral circulation, probably have the power to reduce pulmonary pressure. Some have used them with success in pulmonary hemorrhage. Chloroform depresses the heart and also the respiration and so causes a fall in pulmonary pressure. Aconite likewise causes a lowering of pulmonary pressure. The reason that morphin has been used in pulmonary hemorrhage is because of its lowering influence upon the pulmonary pressure, this being effected largely by slowing the respiration and thus decreasing the amount of blood drawn into the heart from the abdominal vessels. Nevertheless, it has the disadvantage in this form of hemorrhage that with the decreased desire to cough and expectorate, blood is retained in the lung and favors pneumonic infiltration.

Active Congestion

Active congestion is rarely a disease by itself but frequently a part of some underlying infection. In the early stage of pneumonia, tuberculosis or pleurisy, there is local hyperemia. It may be so small or centrally located as not to give definite signs. In patients with high sustained fever such as typhoid, scarlatina, influenza without a frank pneumonia, malaria and other fevers, this condition may be superimposed. Carrière has studied and written concerning this so-called localized acute congestion which he describes as accompanied by chills, fever, cough and expectoration of mucoid or mucopurulent material. The temperature may range between 103° to 104° F. for from three to five days and fall to normal by lysis. This is probably an abortive form of pneumonia caused by an attenuated pneumococcus. While these areas may be in any lobe, they are more frequently found in the lower lobes. Inhalation of irritating gas such as sulphur, chlorin, bromin, etc., is prone to cause general active congestion and may go further to cause bronchitis or pneumonia. The tissue surrounding that portion of the lung compressed by an inflammatory pleural exudate shows a compensatory dilatation of its blood-vessels, but this is probably of little clinical importance. In pulmonary embolus there is a zone of active congestion about the wholly or partially occluded artery, and this frequently leads to diapedesis of the red blood-corpuscles into the surrounding tissue. In streptococcic conditions, erysipelas, cellulitis, etc., the physician should be constantly on the watch for acute congestion of the lungs.

Treatment.—The treatment for such active congestion as implied above is that of the underlying condition. The patient is generally in bed and if not he should be because of the danger of his developing a frank pneumonia. Where there is no acute upper respiratory infection it is not as important to have the air in the room warmed but plenty of fresh air is advisable. There is some difference of opinion regarding counter-irritation of the chest wall, but in general it is favored, and I think of considerable value. Mustard plasters, home-made, are far superior to those purchased, and in the dilution of 1 part mustard to 3 parts flour, will generally produce hyperemia of the skin in fifteen to twenty minutes. Some individuals with tender skin (more frequently blondes) may require a weaker dilution, while others may tolerate without reaction equal parts of flour and mustard. The flour and mustard should be mixed with either lukewarm water or the white of egg, so that the consistency is that of soft butter, and spread between layers of gauze or soft linen and applied to the chest, front and back. To prevent blistering, first cover the skin with petrolatum, sweet oil or salad oil, and to continue the counter-irritation after the removal of the plaster, camphorated oil should be applied. A tablespoon or two of castor oil, disguised in orange juice and a bit of soda if necessary, is an excellent way to clear the intestinal tract. It is generally necessary to follow this up for a couple of days with a mild laxative such as milk of magnesia, to prevent further constipation. In plethoric individuals, venesection may be of value both to relieve congestion and rid the patient of toxins. This should be resorted to only in selected cases of acute congestion but when used 300 to 500 c.c. should be removed. Aconite in the past has been considerably used because of its power to reduce pulmonary pressure, but its depressant heart effect has limited its use. Pain and thoracic oppression are seldom present in mild cases or where pneumonia is not present and so the use of anodynes is seldom required. The mustard plaster is generally sufficient and may be applied thrice daily. It is rarely necessary to stimulate the heart, but where necessary digitalis by mouth, or one of its preparations hypodermically, may be used. For quick but less sustained action, caffein sodium benzoate gr. ii, by mouth or hypodermically, or camphorated oil hypodermically, may be used.

The value of any treatment whether for serious or minor afflictions lies in careful attention to details, and explicit directions to the nurse or family will frequently prove the difference between success or failure even with very commonplace measures.

Passive Congestion

This condition may be acute in onset and of relatively short duration, or it may be very slow and gradual in onset and persist indefinitely. It consists in a damming back of blood into the pulmonary veins either from a high systemic pressure, a weakened and incompetent left heart or a mitral stenosis and insufficiency, presupposing that the right heart maintains fairly well its function. Acute passive congestion is generally the result of straining or exertion and sudden giving out of the left ventricle, and results in so-called cardiac asthma. The back pressure of the blood congests pulmonary veins, venules, capillaries, and, finally, branches of the pulmonary artery. By their swelling and resulting tortuosity the air spaces are encroached upon and more rapid respiration results. With this there is more blood brought to the right heart and thrown into the pulmonary circulation. With this slowing of the circulation, the blood is less well aërated and together with diminished elasticity of the lung tissue, an increased amount of work is thrown on the accessory muscles of respiration, and dyspnea results. This poorly oxygenated blood also stimulates the medulla to more frequent respiration.

From this it will be evident that the symptoms produced are dyspnea, feeling of thoracic oppression, cough with or without clear or bloody mucoid expectoration, cyanosis and weakness. To prevent such an occurrence every means should be taken by the patient suffering from mitral or aortic disease, especially stenosis, arteriosclerosis and hypertension, to avoid undue or sudden exertion. By this is meant hurrying for cars or trains, walking up hill too fast and against a strong wind, overeating or eating too rapidly with the formation of gas, straining at stool, hard coughing spells or repeated sneezing and lifting heavy weights. Overfatigue mentally or physically should also be avoided by periods of relaxation from work and plenty of sleep at night. Frequently a rest period during the day will enable the patient to store up sufficient reserve to prevent such a sudden break in compensation.

Chronic passive congestion may be the prolonged effect of an attack of acute passive congestion where the left ventricle recuperates some of its strength, or it may result from the gradual back pressure of blood in the pulmonary system from any of the above-mentioned causes. The symptoms of passive congestion—dyspnea, cough, expectoration, at times bloody, etc., are more marked when the right heart is strong and prevents a back pressure in the systemic venous system. When this gives out the viscera and extremities become congested and in time edematous. With a chronic passive congestion of the lungs it is not at all uncommon to have infection superimposed because of the diminished resistance of the tissues. This manifests itself by frequent attacks of bronchitis and the "winter cough" of the patient with arteriosclerosis and hypertension is frequently bronchitis and if fever accompanies the condition there is a bronchopneumonia.

Pathologically these lungs are large, heavier than normal, the blood vessels are distended and tortuous, there is a brownish pigment in the interstitial tissue as well as in the epithelium, they are firm and cut with considerable resistance. If the condition has existed for a long time and infection has supervened there will be an overgrowth of fibrous tissue especially in the lower lobes.

Treatment.—The two chief standbys in treatment are rest and digitalis—in other words, the treatment of the underlying cause. Where the condition is severe when first seen, massive doses of digitalis—4 to 6 drams in twenty-four hours—and then, minims xx to minims xxx, four times daily, are required gradually reducing the dose as progress takes place and the compensation is restored. Rest, in many instances, must be absolute even to the point of avoiding talking or self-feeding. Some of the congestion, especially if infection is superimposed, may be relieved by counter-irritation—mustard plasters. Elimination by at least one or two full soft stools daily and hot packs to the body if the systemic blood-pressure is elevated, and a light diet with especial restriction in salt, will be of help. Where the pressure is high, and particularly where the patient is plethoric and cyanotic, venesection of 400 to 500 c.c., may be the deciding factor in saving life. Camphor and caffein, alternating with digitalis preparations (mouth or hypodermically), are at times very useful.

As passive congestion, when severe, closely borders on pulmonary edema, the treatment in more extreme cases will be described there.

Hypostatic Congestion and Pneumonia

By hypostatic congestion is meant the gravitation to the dependent portions of the lungs of the congestion and edema which develops in elderly persons or cachetic individuals suffering some severe acute illness or one of long duration where it is difficult to change their position. Particularly is this to be feared where old persons have broken a leg or hip. The avoidance of a hypostatic congestion is generally of more importance than strict carrying out of all the best methods of immobilization of the leg. Where the general resistance to disease is lessened by age and injury, infection is very prone to creep in on the hypostatic congestion, causing hypostatic pneumonia. The symptoms consist in gradual onset of cough, mucoid or purulent expectoration and fever.

Physical signs increase from slight dulness and râles at the bases or other dependent portions of the lungs to lobular or lobar distribution of bronchial breathing and increased vocal and whispered fremitus.

Treatment.—The treatment consists in frequent change of position, from side to side, and the assumption of the sitting position for as much of the twenty-four hours as possible. Stimulation, as above described, is generally necessary for the heart and the treatment of the lungs is identical with that of bronchopneumonia or lobar pneumonia (see Chapter XXIX, Vol. II).

Pulmonary Edema

This is a very important subject and deserves much more attention than has been given to it. Apparently there are two types of pulmonary edema, one of which is of gradual onset and follows slowly but steadily in the train of chronic passive congestion, while the other is frequently termed "superacute pulmonary edema," because of its very abrupt onset, frequently with no premonitory symptoms, except at times preceding attacks.

Etiology.—In the group of chronic passive congestion cases, there is little to add either as to cause or progress and its treatment will be discussed together with that of the "superacute" condition. In this later form there is generally the history of hypertension involving the cardiorenal complex—dyspnea on exertion, slight cough, occasional headaches, possible disturbance of vision, congestion of the varices—nasal, esophageal, hemorrhoidal—with bleeding at times, and nocturia with the typical urinary finding of renal arteriosclerosis, low specific gravity, a very slight trace of albumin, or none at all, and a very few hyaline casts. Mitral stenosis with acute auricular fibrillation has been cited as a not infrequent cause of the acute form. Another cause is mediastinal or pulmonary tumors which are so situated that violent coughing spells or a strain causes such a severe congestion that edema results. Again a severe fibrosis of the lung which is the result of too intensive deep ray therapy of lung tumors may rather abruptly cause a pulmonary edema of fatal course. It is not uncommon to find a zone of edema about the inflammatory process in pneumonia, tuberculosis or pleurisy and in such other acute infections as scarlet fever, typhoid, rheumatism and influenza. This requires very little attention outside the original or primary cause. By the rapid withdrawal of comparatively large amounts of pleural fluid (1,200 to 2,000 c.c.), which has been present for some time, an acute pulmonary edema may result. The explanation for this probably lies in the fact that a portion of the lung, collapsed for some time, is less elastic and the blood-vessels are constricted. By this sudden re-opening and dilatation of these vessels an edema results. Riesman has termed it "congestion by recoil," and another explanatory phrase is "edema ex vacuo." This form of edema may be avoided by the removal of not more than 1,000 c.c. at least at the first time and this through a small trocar and slowly. Another type of acute pulmonary edema is that which results from mechanical pressure on the vagus. The following case will illustrate:

"A child, age 8, suffering from cervical Pott's Disease, became restless one evening from no particular cause. In his movements, he lost the good position in which his spine was held by a split plaster jacket. To make him more comfortable, the nurse, without appreciating the danger incurred, removed his plaster shell and very promptly the boy developed an alarming pulmonary edema and within 20 to 30 minutes he seemed almost moribund. Following no result from morphin and atropin, as a last resort we replaced him in his jacket shell and within a few minutes, the edema cleared. Next morning he was happily reading his piece of the Sunday paper as if nothing had happened."

Insufficiency of the coronary arteries, especially those of the left ventricle, may be the direct cause for the left heart giving way and not being able to keep up with the task given it by the right heart.

Theories as to Pathology.—(1) Welch's explanation as to why this form of superacute pulmonary edema takes place is that there is a sudden inequality in the work done by the right and left heart, the left doing only half as much as the right. This severe inequality shortly leads to such passive congestion that edema takes place. (2) And for this there may be an increased permeability of the vessels' walls. This again brings up the question as to whether there are or are not vasoconstrictor and vasodilator nerves governing the lung capillaries. Although it has not definitely been proved that there are, it seems that branches of the vagus do control the action of these vessels. Kraus found that severe pulmonary edema took place in rabbits that were injected intravenously with 200 to 300 c.c. of normal saline and their vagi cut. (3) That the vagus and adrenals also play a part in this edema is evident from the work of Auer and Gates. Their rabbits developed marked pulmonary edema after the injection of adrenalin intratracheally and the cutting of the vagi. Without this latter procedure, only very slight edema was noted. As adrenalin intratracheally exerts a bronchoconstrictor effect, the aspiratory or cupping influence of each inspiration tends to draw fluid from the vessels into the air spaces. With artificial respiration they were able to diminish the amount of pulmonary edema on the ground of overcoming this condition. The success, too, of atropin is therapeutic verification of this theory. Stenosis of the trachea in these adrenalin rabbits with vagi intact tended to produce pulmonary edema. They also observed the inequality of the right and left heart action described by Welch. Laignel-Lavastine and Jelliffe and White feel that it is a vegetative syndrome indicative of endocrine disorder, the former suggesting adrenal insufficiency, while the latter at least tentatively holds that "it is a neural reaction brought about by the action of the vegetative nervous system, which controls reciprocal tension relations or cellular chemical composition relations."

Symptoms and Physical Signs.—The acute or "superacute" form frequently occurs during the night and the patient is awakened by dyspnea, leading to orthopnea, a distressing dry cough leading to a loose cough with very large amounts of pink frothy sputum, which may well out of his mouth and drown him if the condition is not relieved. An intense feeling of thoracic oppression is evident, a very anxious expression and cyanosis with later a grey pallor. On examining the chest, it is impossible to percuss the cardiac borders because of compensatory emphysema; there is labored breathing with dulness extending up from the bases for varying distances, sometimes one-half the extent of the lung, and over this there is distant breathing and a variable number of coarse, moist indeterminate râles. The pulse is generally rapid and full. It is difficult at times to differentiate the signs at the bases from pleural effusion which is present, too, at times.

Preventive Treatment.—The avoidance of any form of overstrain in patients with cardiac or renal disease is extremely valuable. Care as to diet, avoiding indigestable foods, condiments, including especially salt, raw fruits and raw vegetables (gas producers), eating slowly, avoiding straining at stool, and also helping elimination by two full soft stools daily are essentials; digitalization of the patient if break in compensation is threatening and the use of hot packs and nitroglycerin or the nitrites where the blood-pressure is excessive. For the severe acute attacks, morphin, gr. ¼; atropin, gr. 1/150, and nitroglycerin, gr. 1/100, are indicated, the first because of its effect on the mental strain as well as to diminish the flow of large quantities of blood from the abdominal veins, brought on by dyspnea, the atropin because of its relaxer effect on the bronchial muscles and for the absorption of fluids and the nitroglycerin for lower systemic blood-pressure. Amyl nitrite pearls will tend indirectly to diminish pulmonary pressure and act quickly. Venesection, 500 c.c. at once, has been the deciding factor, I feel, in two cases that have come to my attention, but if not associated with high systemic pressure this is contra-indicated. The removal of this blood not only diminishes pressure but also toxins and prevents further inundation of unaffected lung tissue. Catheterization of the lung through a tracheotomy opening and aspiration of the fluid has been suggested. For the bronchoplegia, strychnin has been advised and electrization over the vagi has offered good results in some cases. Teissier, of Lyons, has used carbonic acid, rectally, in one case with success, "due to the stimulating effect of the gas on the respiratory center." In extreme cases, where venesection has failed, aspiration of blood from the right auricle by puncture in the fourth right interspace may give relief. Iodids are said to predispose to pulmonary edema and their use should be avoided where conditions favor the occurrence of edema.[1]

The after treatment should be dietary, rest, digitalization, theobromin gr. x-xv t. i. d., p. c., and the prophylactic treatment already suggested.

---

[1] Riesman has reported good results from artificial respiration.

Pulmonary Thrombosis and Embolism

Pulmonary thrombosis is less common than embolism but may be present in the arteries, veins or capillaries and may be due to changes in the composition of the blood, slowing of the rate through the lungs as in passive congestion, trauma—a rare condition with the pulmonary vessels but sometimes encountered when chemical irritants or foreign bodies are inhaled or trauma crushes the thorax. A small embolus which does not entirely occlude an artery may locally slow the circulation and by damage to the intima predispose to thrombosis. Thrombosis in the capillaries or veins may lead to systemic emboli, a condition probably more frequently encountered in pneumonia than recognized. Pleural effusion is not an uncommon cause of thrombosis and systemic embolism.

Pulmonary emboli arise most frequently in the veins of the lower extremities or pelvis, in endocarditis or from mural thrombi in the right heart, either in the auricular appendage or between the columnæ carneæ. They may be single or multiple, small, medium or large. Predisposing diseases, in which thrombi may occur and thus lead to emboli, are typhoid or other infectious forms of phlebitis; postpartum thrombi in uterine veins, thrombosed vessels in the wound of a laparotomy incision, the vessels presenting no evidence of infection and there being no cause for such a condition unless it be trauma; crushing injury where considerable tissue is bruised, either external tissue or traumatized viscera. Jugular thrombosis from mastoiditis may give rise to infected pulmonary emboli. As above mentioned, an acute vegetative endocarditis of the tricuspid valve, especially where this is combined with hypertrophy and dilatation, together with the thrombi in the right heart may give rise to frequent showers of emboli. Thrombi in chlorotic individuals are more prone to cause emboli than in others because of the brittleness of the clot.

Special mention should be made of fat emboli for they are not uncommon, accompanying fractures of long bones. Here the fat is mobilized and gains entrance to the veins by injury from spicules of the broken bone. This is particularly true of fractures in elderly people where there is proportionately more marrow. The fat sometimes makes its way through the pulmonary artery and capillaries and is found in the peripheral circulation. It seldom causes the platelet thrombosis seen where blood-clots lodge in the pulmonary artery. Symptoms of fat emboli may not appear for some time (one day to two weeks) after they gain entrance to the lung. If large they may cause the same symptoms as thrombotic emboli.

Air emboli are occasionally seen but danger only results from such air if it is in several cubic centimeter amounts. Because of the relatively large negative pressure in the jugular vein this is the point from which many arise. As the air is drawn in, a suction sound is generally heard. Death is due to a paralysis of the right heart due perhaps to its endeavor to compress a substance less resistant than blood. The right ventricle usually contains only a mass of froth. If the air passes the heart it may cause local congestion by pressure in the capillaries. I have observed air embolism in one case of attempted artificial pneumonothorax, where it was difficult to find the pleural cavity. With a small oscillation of the manometer a little air was allowed to enter, following which, in a few seconds, collapse occurred. The patient seemed moribund but with cardiac stimulation he recovered and showed a right facial and upper extremity paresis for about three days which completely disappeared (air must have been introduced into a pulmonary vein).

Oil emboli are occasionally encountered as the result of the injection of oily suspensions of mercury, camphor, etc., giving rise to small pulmonary infarctions because of the irritant action of the drug. Precaution should always be exercised by withdrawing the piston of the syringe when in place before giving such medication.

Large Emboli.—The symptoms of large emboli are thoracic oppression and dyspnea, distress, as contrasted with the pain of angina, pallor and then cyanosis, cough and sometimes bloody sputum. The pulse becomes rapid and thready, the pupils are dilated, the skin is covered with a cold sweat and at times a rough murmur is heard over the left second interspace or third rib near the sternum, which is coincident with systole and extends beyond it. This was described by Litten and he ascribed it to a narrowing of the blood stream. Death generally supervenes within a few seconds but may be delayed for hours.

Treatment.—In those who survive, rest is very essential and morphin, gr. 1/4, should be given and repeated in thirty minutes if necessary. Because of the possibility of its being a rider embolus, any motion, even the exertion of talking or eating, should be avoided. Trendelenberg has twice opened the pulmonary artery and removed the clot but in each instance death ensued. If the patient survives the immediate effects, strict immobilization of any phlebitic area may prevent further dislodgment of thrombi. As these emboli may occur at night, during sleep, or while changing from a reclining to sitting posture or slight exertion, these and any other strain should be avoided in an individual with marked thrombophlebitis. Venesection in desperate cases may tide the patient over a crisis by reducing pulmonary pressure.

Small Emboli.—Small emboli may give such trivial symptoms that they may be disregarded by the patient, or if larger may simulate small areas of spontaneous pneumonia. Just as the large emboli come from a fully developed thrombus, generally recognized and treated, so the smaller emboli frequently come very early in the process of phlebitis and often days before the disease is recognized. Several small emboli may come from along the path between the distal and proximal thrombi and may present a cycle of showers. Whenever the patient is suffering from any of the above-mentioned predisposing diseases, the possibility of emboli should always be borne in mind and avoided by long-continued rest of the part, frequently six weeks or more. Either active or passive motion or massage may provoke a shower of small emboli if organization of the thrombus is not complete.

Symptoms.—The symptoms of smaller emboli are pain referred frequently to the axilla, shoulder or diaphragmatic region, and present on breathing; cough is generally present and there may be blood in the sputum. When present it is a helpful aid in making the diagnosis. It may be bright or prune-colored but practically never rusty. There may or may not be any dyspnea, depending upon the size of the embolus. The temperature is elevated and may remain at 103° to 104° F. for three or four days. Physical examination in very small emboli reveals no signs because of the collateral circulation including the branches of the bronchial artery. When larger fragments are thrown into the circulation we find localized dulness and diminished breathing. Pleuritic crepitations are frequently heard and later bronchial breathing and a few fine crepitant râles. As the base of the infarct is on the pleura, it is safe to assume that the area observed is the maximum that is involved. Diminished tactile fremitus may lead to the suspicion of fluid in the pleural cavity but generally none is found at aspiration.

Emboli, small and large, are generally located in the right lung rather than the left because of the larger artery leading to it, and in the lower lobe more frequently than in the others, this being partly aided by gravity. Infarction is to be differentiated from diffuse bronchopneumonia and lobar pneumonia in which a whole lobe is involved because of its discrete localization. Tuberculosis more frequently involves the upper lobes and is ushered in by a history of preceding malaise, etc., in contrast to the suddenness in embolism. Lung tumors may show discrete outline but are of gradual onset and without fever.

Prognosis.—This depends on the size of the embolus and the preexisting condition of the lungs. Passive congestion or infection leads to septic infarction as does an embolus of infected material. Where the conditions are unfavorable a lung abscess may develop and lead to serious consequences. Where the circulation is normal and there is no infection the infarction clears within a few days. In elderly people, or those suffering from any wasting disease, their general low resistance may convert a favorable to an unfavorable prognosis.

Treatment.—The treatment of these smaller or medium-sized emboli is that of pneumonia: rest, fresh air, avoidance of all exertion which may dislodge other thrombi, local counterirritation over the involved lung area by mustard plasters, thus frequently relieving congestion and distress.

Morphin may be necessary but if so, small doses of 1/8 gr. to 1/6 gr. usually are sufficient. Codein, gr. 1/4, by mouth is usually satisfactory. Cardiac stimulation may be necessary if there is much toxemia.

BRONCHOPNEUMONIA

PHILIP H. PIERSON

Bronchopneumonia is generally a disease of childhood or late adult life but during epidemics, such as influenza or measles, it seems most virulent among those in middle life. Clinically, acute infections which attack the upper respiratory tract and gradually make their way down through the trachea and larger bronchi into the bronchioles, and thus into the center of a lobule, present no sharp line of distinction, the one merging into the other gradually, and thereby the infection called bronchopneumonia is differentiated more by degree of reaction than by specific physical signs. In bronchitis where the temperature persists more than two or three days above 102° F., we must consider that there is a more or less lobular arrangement to the infection. Somewhat different was the influenza bronchopneumonia in that it was frequently found during a relapse and progressed with marked rapidity. In this type, the organisms (for the exact causative agent is generally believed to be several and not one type of organism) were introduced through the respiratory tract, spread downward into the trachea and into the bronchial divisions by extension from the original source, this being aided by a bloody extravasation into the air spaces. In different localities slightly different pathological distributions were encountered as were also variations in the predominating bacteria. MacCallum has written a very complete and clear description of the pathology and suffice it to say here there was marked swelling of the bronchial walls as well as those of the smaller air passages, with hemorrhage into the bronchioles and dilatation of the alveoli. The firmness of these areas made them stand out as subpleural nodules, in some instances discrete, but generally so confluent as to simulate lobar pneumonia. The marked interstitial infection was largely responsible for the fibrous reaction and loss of normal tone which later in many cases resulted in localized emphysema or chronic pneumonia. Honeij states that during the early epidemics, bronchopneumonia occurred in 5 to 10 per cent of the cases of influenza and of these there was a mortality of 60 to 70 per cent. It is probable that the part played by the influenzal infection was one of acutely lowering the resistance of the patient so that the streptococcus or pneumonococcus quickly caused either the acute hemorrhagic pneumonitis described by Selby or the confluent lobular types described by others. The assembling of a large number of men from rural and urban surroundings and the more or less crowded quarters in the training camps predisposed to epidemics of measles, and, with the superimposed streptococcus infection, bronchopneumonia was soon in epidemic form.

Symptoms.—The symptoms of bronchopneumonia are generally of gradual onset, consisting in a steadily increasing fever accompanied by rise in pulse and respiration, a tight, constricted feeling throughout the chest, especially under the sternum, with a dry rasping cough later becoming productive. Where the involvement of lung tissue is extensive, cyanosis is generally present. In children and infants the onset is much more acute, while in elderly people it is more protracted. The average duration of the acute symptoms is about seven to ten days. As onset is gradual so recovery is by lysis, and relapses are not uncommon.

Prophylaxis.—In children and elderly people, lack of proper nutrition is a potent factor in causing bronchopneumonia and special care should be exercised in these individuals to avoid respiratory infections. Becoming overtired or fatigued mentally or physically, exposure to cold, intercurrent infections, such as measles, scarlet fever, diphtheria, typhoid or chronic digestive disturbance, lower the individual's resistance. Frequent "colds" in children are often caused by wrong clothing, some parents overclothing and others allowing too much exposure. The proper method of clothing during the cold season should be to cover the arms and legs completely, the use of a single shirt and waist, the former being preferably of light-weight wool, and long drawers of the same light-weight material. This will not lead to perspiring while indoors but, by the addition of suitable overcoat or wraps, the cold of the outdoors will not be detrimental. A frequent cause for acute upper respiratory infections is diseased tonsils, adenoids or sinuses and these should not be allowed constantly to infect the individual and lower his resistance. A few gymnastic exercises on arising following by a cold bath is very helpful in preventing respiratory infections. These baths are tolerated more easily if the room is warm and the person stands in warm water. Proper simple food at regular hours and plenty of rest at night are important measures to prevent infections.

Overcrowding is a very necessary thing to avoid, especially in overheated rooms. In hospitals patients suffering from bronchopneumonia should be isolated, preferably in single rooms, and if this is impossible at least in cubicles, with special attention to the disinfection of the nurse's and attendant's hands and burning of the sputum. During the influenza epidemic, face masks consisting of several layers of gauze covering the nose and mouth were in general believed to be a distinct help in preventing the spread of droplet infection. I have seen it used by nose and throat specialists as a personal precaution and it might well be applied to dentists. During the epidemics of acute respiratory diseases, places of amusement such as theaters and crowded department stores should be avoided. The good physical condition of the individual should again be emphasized as an important preventive measure. He will be much less liable to infection if he eats simply and regularly, gets plenty of sleep and pays particular attention to his bowels. Mouth hygiene, too, is extremely important, brushing the teeth two or three times daily and rinsing the mouth with the following solution: Dobell's solution, 1 part; extract of witch hazel, 3 parts; water, 4 parts.

Prophylactic Vaccination.—The experience of army and navy surgeons, not only in this country but abroad during the influenza epidemics, gave promise that a series of prophylactic injections reduced both the morbidity and mortality from respiratory infections in general and pneumonia in particular. They were able to use a large number of men for treatment and control, and felt that this measure reduced morbidity from pneumonia at least one-third, and acute respiratory infections, including pneumonia, one-fifth. Some authors were even more optimistic. The vaccine used by Duckwall (B. F.), was prepared from Types I, II and III in saline solution, there being 1,000 organisms per cubic centimeter. His first injection consisted in 0.5 c.c., the second seven to ten days later was 1 c.c. and the third injection of 1 c.c. was also given seven to ten days later. Some vaccines have been made of the pneumococcus, plus strains of streptococcus, influenza and other bacteria and have been helpful in preventing the frequent respiratory infections which lead to bronchopneumonia in certain individuals. Before this method is used, except during epidemics where there is immediate need for protection, foci of infection should be removed if the result is to be at all lasting. (This subject is more fully discussed in Vol. III, Ch. XIV.)

Specific Treatment.—Within the last few years, progress has been made in the treatment of pneumonias caused by Types I and II pneumococcus, especially Type I. This organism may be found in bronchopneumonia, although frequently in combination with streptococci and other organisms, and so a brief statement of the serum treatment will be outlined, although for a fuller description reference should be made to the chapter on Lobar Pneumonia, or to the work of Cole. It is important to determine the type of pneumonia as early as possible and most cities are now able to do this within five hours by the Avery method. As previously stated, Type I has given the best results although some are using the serum prepared by using both I and II. The serum is prepared by inoculating a horse with first killed and then live pneumococci. The potency of this serum is such that "0.2 c.c. of the serum will regularly protect a mouse against at least 0.1 c.c. of an 18-hour broth culture of pneumococcus of such virulence that 0.000001 c.c. of the culture will kill a mouse of 20-gm. weight within 48 hours.

"To be of the most value the serum should be administered within

48 hours of the onset of the disease. The first dose is 90 to 100 c.c. of the serum intravenously, repeated every 8 hours until there is a fall of temperature and abatement of symptoms. The serum should be at body temperature and administered slowly. The average total amount necessary is 200 to 300 c.c. but severe cases may require 600 c.c. The effectiveness of this treatment will be manifested by (1) sterilization of the blood, (2) arrest of the progress of the local lesion, (3) amelioration of subjective and objective symptoms and (4) decrease in the mortality of the disease. There may be three types of serum reactions: anaphylactic shock, which may take place where large amounts of serum are administered by any method; thermal reaction, occurring about 20 minutes after the injection and generally not serious; and serum sickness present in about 50 per cent of the cases and generally mild." Cole has recommended desensitization of patients who are at all asthmatic or whose reaction to 0.2 c.c. of a 1:10 dilution of horse serum intradermally amounts to a wheal 1½ inches in diameter within 5 minutes. This may be accomplished in mild cases by the subcutaneous injection of 0.5 to 1 c.c. of serum a few hours before the treatment begins. In more sensitive cases it is necessary to begin subcutaneously with 0.025 c.c. of the serum, doubling the dose every half hour till 1 c.c. is given, then intravenous injection of 0.1 c.c., doubling the dose every half hour until 25 c.c. are given. Four hours later 50 c.c. may be given. After 8 hours, the treatment may be continued in the regular manner. To combat the anaphylactic reaction be prepared to give ♏ x of 1:1,000 suprarenal extract. It is claimed on good authority that the mortality is reduced from 25 per cent to 30 per cent in untreated cases to 9 per cent to 10 per cent in the treated cases.

Leukocytic extract prepared from the blood of normal animals has proved of considerable value in some instances. Theoretically, it is supposed to increase the number of leukocytes in the blood and to increase the phagocytic and ferment splitting power of the individual polymorphonuclear leukocyte. Sometimes both of these actions are observed but more frequently the latter. Numerous white counts have failed to reveal any marked increase in these cells, but in my experience prompt amelioration of symptoms and pathology in many cases substantiates the latter claim. In adults 2 c.c. may be given daily, while in children 1 c.c. should be the dose.

Quinin and urea hydrochlorid (soluble) in doses of xv to xxv gr. two to four times daily, subcutaneously, has been recommended by Solis-Cohen, and used with considerable enthusiasm for many years by other observers. It cannot be used in children, but appropriate doses of euquinin may be. Quinin muriate in doses of gr. v to x, three or four times daily may be of value.

The *iodids* have been recommended especially during resolution in those suffering from lues. Too large doses or doses continued over too long a period in non-luetic individuals probably keep up excessive secretion and delay resolution.

Creosote formerly enjoyed considerable favor but in recent years has not been so popular. It has been used in hypodermic form as "creosota pura" in ♏x to ♏xv doses or by mouth as creosote carbonate in ♏iii to ♏v doses.

Glucose has been recommended as an adjunct in the treatment of pneumonia. H. J. John uses about 250 c.c. of a 10 per cent solution and frequently combines such other therapy as antipneumonic, streptococcic, or tetanic serum with it as well as digitalis, morphin and atropin. Nourishment and better elimination is the claim for its use.

Streptococcic vaccines have been used by some with limited success.

Hygiene.—Promising as specific therapy may be, careful attention to hygienic principles is absolutely essential. It is always better to go into too explicit detail than to allow too much for the patient, his friends or nurse to surmise and carry out in a less advantageous manner. The temperature of the room should be kept as nearly 65° F. as possible and this is accomplished by the use of an open fireplace providing heat and ventilation, or an electric stove upon which is placed a pan of water. This moistens the air and prevents the drying of the mouth which is so annoying and which also predisposes to oral sepsis. Tincture of benzoin, a dram to the pint of water, as an inhalation is soothing to the mucous membranes. If the room becomes too cold, too warm or too dry, the patient is generally less comfortable. Of course, the patient should be in bed, but in a semi-reclining posture for at least several hours each day. Change of position from side to side is very important, where there is naturally congestion, and this is particularly essential in elderly persons. The terminal bronchopneumonia following a fractured femur in old people is so common an example of hypostasis that it should be avoided even to the point of allowing such an individual to be sitting up and pay less attention to the strict immobilization of the fracture. It has been said that one-third to one-half the treatment of pneumonia lies in the nursing, and attention to the change of position is one of the important nursing duties. The patient should be protected from drafts for there is frequently perspiring, and chilling may easily lower the patient's resistance. This should not in any way limit the amount of fresh air supplied the patient for this is one of his chief forms of nourishment. The mouth should receive constant attention because of the discomfort from becoming dry due to so much mouth breathing, and because infection about the teeth or particles of food may be inspired and set up new foci of infection in the lungs. After cleansing with peroxid, liquid petrolatum may be applied to lips and tongue, thus keeping the tissues constantly soft. The diet should be liquid or soft solid during the height of the fever, taking care to avoid any indigestible foods, and to combine high caloric value with each feeding, milk with cream, eggnogs, ice cream, gruels, milk toast, soft-boiled eggs, prune whip, fruit syrups with rich milk, cream soups, etc., are examples of nourishing feedings. Forcing fluids is a most excellent method of reducing toxemia and sparing the kidneys. For this orange juice in the proportion of 8 ounces of juice to 12 of water with 3 tablespoonsful of milk sugar, combines a cooling drink with some nourishment. Other fruit juices may be used. Where there is no edema fluids should be forced so that the "measured and recorded" urine output is 60 to 90 ounces per day. This measured output is a definite gage as to the amount of intake necessary. Mustard plasters, although criticised and thought useless by some, in general, are very pleasing to the patient, for a good reaction does relieve much of the congestion and oppression. Pain is also so considerably relieved that opiates are sometimes unnecessary. The plaster should be made by mixing 1 part of mustard with 3 parts of flour, using either lukewarm water or the white of egg to mix it into a workable paste. This is spread between gauze, making a plaster 5 by 9 inches. First apply sweet oil or vaselin to the chest and then place one of these plasters on the front and one on the back of the chest. They should remain on till the skin is pink, generally fifteen to twenty minutes, and on their removal either camphorated oil or the following liniment may be applied: Ol. sinapis dram ss, Ol. gaultheriæ oz. ss, Ol. olivæ q. s. ad. oz. iv. This counterirritation and heat may well be kept in by a thin piece of flannel next to the skin. This is not to be left on indefinitely but until the patient is able to be up and about. Mustard compresses if properly applied may be of more value especially in children. A basin of mustard water, 1 tablespoonful to the gallon, is kept hot in such close proximity to the patient that large towels can be wrung out of this and wrapped around the chest, and this in turn covered by a piece of rubber silk or a small blanket. These should be changed frequently enough so that the skin shows a good pink glow at the end of ten minutes. This may be applied for twenty to thirty minutes, three or four times daily. Cold compresses on the chest are enjoyed by some, made by wringing out a piece of flannel and laying over the chest and covering it with several layers of dry flannel. These are called Priessnitz applications.

Fever.—In bronchopneumonia this is generally not as constant a factor as in the lobar type, but for temperature over 103.5° F. a warm bath with the water temperature 103° F. is very restful and quieting. The patient should not be exposed too much but one member may be sponged and dried at a time. Debilitated patients do not tolerate cold sponges. If plenty of friction is used in any bath the skin reaction, superficial dilatation, will reduce the temperature. An ice-cap to the head will often reduce the delirium which is present in some instances even before the fever has reached 103° F. Forcing the fluids, as suggested above, will generally ward off much of the toxemia which otherwise would exist. When nausea prevents forcing fluids, the Murphy drip may be necessary and should be used before toxic symptoms become too manifest.

Cyanosis and Dyspnea.—These may be of two origins, cardiac insufficiency or lack of sufficient aërating surface in the lung. This latter is the result of the congestion and edema together with smaller or larger areas of atelectasis. The mustard plasters or compresses, as mentioned above, will help to withdraw some of the pulmonary congestion and edema. Atelectasis may be due to postural predisposition not to expand certain parts of the lung or it may be due to plugs of mucus or exudate obstructing the lumen of a bronchiole. Change of position, relief of distention and expectorants, may be of great help in relieving this condition. Ammonium chlorid, carbonate or muriate in gr. v doses in combination with a syrup is a good expectorant. Potassium iodid has been mentioned including its advantages and disadvantages. Atelectasis in children is often relieved by seating them in a tub of warm water for several minutes and pouring a glass or two of cold water over them, thus causing them to take a big breath.

Cyanosis is *prima facie* evidence of lack of the normal amount of oxygen in the blood. It is only since the World War that any considerable use of oxygen, as a therapeutic measure, has been made and this originated in the work with those afflicted with either acute or chronic gas poisoning. Methods have been devised for estimating the oxygen saturation of both arterial and venous blood and, by comparison with the normal, the deficiency is more easily noted and the effect of treatment gaged. Since then oxygen has been used in the treatment of both lobar and bronchopneumonia by one of several forms of apparatus. Haldane devised a face mask surrounded by a pneumatic rubber rim. In this mask is an opening for the ingress and egress of air but this aperture offers just sufficient resistance to allow, with each inspiration, the opening of the valve leading to a rubber bag which receives a steady stream of oxygen at the rate of 1 to 4 liters per minute. Distal to the rubber bag is the oxygen tank with pressure gage and reducing valve, allowing a constant flow of the gas till the tank is empty. If the mask is not held tightly to the face oxygen will be wasted. This was the chief disadvantage of the old funnel method. The reinspired air in the mask, being rich in CO_2, tends to stimulate respiration and this is of advantage because the air is also rich in oxygen. To some this mask is uncomfortable and Barach and Woodwell devised a modification, using the mouthpiece of a Benedict respirator. This left the nose exposed and, as breathing during pneumonia tends to be by mouth, with the added request to breathe this way, it was estimated that the patient received about 50 per cent oxygen. They interposed a jar of soda lime between the rubber bag and the mouthpiece, thus withholding all CO_2 and allowing only oxygen to be breathed back and forth into the bag. The oxygen was fed into this bag as in the apparatus of Haldane. In the later apparatus there was no rebreathing of the oxygen as in that of Barach and Woodwell. By using either of these apparatuses for thirty minutes to two hours, marked relief from cyanosis and some slowing of the pulse has been noticed frequently. By their repeated use lasting benefit has been observed. Meakin reports that with the Haldane apparatus used for several hours in a patient with pneumonia the unsaturated oxygen content of the blood was reduced from 15 per cent to 17 per cent (normal 4 per cent to 5 per cent) to 3 per cent. There has been some difference of opinion as to whether the anoxemia is due to the rapid shallow breathing typical of this disease (Meakin), or whether it is due to diminished absorbing surface (Stadie). Stadie has devised an oxygen room, where a 50 per cent saturation may be kept up for long periods (sixteen hours a day) and in which proper nursing and nourishment may be constantly supplied. This has a distinct value in hospitals both for pneumonia and certain heart conditions. The oxygen bed-tent of Hill has some advantages. This allows oxygen to be present at certain approximate percentages. The CO_2 is absorbed and the air is allowed to enter and leave the tent. Nasal catheters have been used for oxygen administration but are of less value than any of the preceding devices. Oxygen therapy certainly has a place in pneumonia both for the relief of such symptoms as cyanosis and tachycardia and for the avoidance of oxygen starvation of brain cells, heart muscle and other organs which may be a more lasting danger.

Distention.—Distention is frequently a very difficult condition to combat. "Gas" enemata consisting of 2 ounces each of saturated solution of epsom salts, glycerin and water, retained, plain glycerin, or milk and molasses (1 part molasses, 2 parts milk) are very effective. Combined with these, turpentine stupes (wring out flannel cloths in 1 dram turpentine to 1 pint hot water and apply over abdomen which is first covered with oil) and a rectal tube may be used to advantage. Where these fail, pituitrin solution 1 c.c. hypodermically is often of value in stimulating the contraction of the smooth muscle.

Circulatory Collapse.—Where bronchopneumonia seems imminent, many clinicians believe that the administration of digitalis, before the heart has been subjected to the toxemia, is preferable to waiting until there is an urgent demand for its use. As it takes twenty-four to forty-eight hours to obtain the benefit of the drug after its use is begun, preparedness is of great value. When nausea is present any of several hypodermic preparations may be used. It is always best for the clinician to use the preparation to which he is most accustomed, whether it be a standard tincture, the leaves, digalen, digitalin or other forms.[1] If digitalis has not been used, strophanthin may be used intravenously in an emergency. Caffein sodium benzoate in gr. i or ii doses depending upon the age, alternating with 1 to 2 c.c. of a 10 per cent solution of camphor in olive oil, hypodermically, will frequently hold the pulse and blood-pressure over a short period of impending collapse. Strychnin gr. 1/30 to 1/60 is a general stimulant and secondarily aids the heart. Whisky was and still is considered indispensable by some in the management of the circulatory support during pneumonia, but the consensus of opinion is that occasionally there may be need for its food and stimulation value over a few days, especially when it is impossible to get sufficient nourishment into the patient. As its action is at first stimulating and then depressing, and because there is some irritation of the kidney in its excretion if not fully burned, it is not to be made a routine measure but held in abeyance until its full effect is desired. Of course, in the management of a patient accustomed to large quantities when well, it is necessary to give it, for otherwise delirium tremens might develop. When needed a half ounce every three to six hours as a hot toddy is sufficient. In similar circumstances, with children, sherry may be used. Venesection may be resorted to where cardiac failure is imminent, 300 to 500 c.c. being withdrawn.

---

[1] Digitalin is so markedly inferior to the other standard preparations that it should not be used.—Editor.

Adrenalin ♏ vii to x, hypodermically, may be used where blood-pressure falls too low but the effect of small doses should be studied before full doses are used. With some the acceleration of the pulse is a decided disadvantage.

Pain or cough when it interferes with sleep is an indication for small doses of morphin, gr. 1/8 to 1/6, or codein, gr. 1/2, by mouth or hypodermically repeated frequently if necessary. During the daytime if these symptoms are not severe opiates should rarely be given because of their tendency to reduce the desire to expectorate. With a good night's rest, the patient can fight his disease with much more strength during the day. A chest binder is a great relief for pain, for with a rasping cough the support given is satisfactory. Adhesive strapping may be resorted to if pleurisy becomes severe, but its irritant action on the skin and its obstruction to counterirritation are disadvantages. An expectorant of ammonium carbonate, gr. iii to gr. v, or fluid extract of grindelia (1 to 2 drams) helps to loosen the secretions, and facilitate expectoration.

Complications.—The most frequent complications are interlobar pleurisy, small pleural effusions, sterile or infected, pericardial or mediastinal effusion, otitis media, meningismus or meningitis, and pyelitis. They should be treated in the same manner as when they occur independent of pneumonia. When an unexplained fever arises do not fail to be on the lookout for some such infection, especially when the patient is too sick to recognize or locate his trouble. Lung abscess or gangrene may develop. Tuberculosis may be reactivated by the severe reaction within the lungs. Diarrhea, if severe, may require deodorized tincture of opium in spite of the fact that its slightly depressing action is disadvantageous. The delirium which at times suddenly develops requires attentive nursing to prevent serious harm from exposure or suicidal attempt.

Convalescence.—Care should be exercised that the patient does not return to regular duties too soon. Rest of the fatigued heart may take weeks, perhaps not always in bed, but with such gradual increase in exercise that lasting damage to the myocardium will not result. Good food, plenty of sleep, and if possible a trip to a temperate climate where continued outdoor life may be enjoyed, will help the process of repair. Tonics of iron or arsenic are necessary in some cases.

CHRONIC PNEUMONIA

Philip H. Pierson

To understand clearly chronic pneumonia, it is necessary to have a definition of pneumonia. Pathologically, pneumonia is an infectious disease, with either an acute or gradual onset, producing consolidation of parenchymatous lung tissue generally in lobar or lobular distribution, the latter at times becoming confluent and simulating clinically the former. In general, pneumococci are the causative agent in lobar pneumonia while streptococci are the cause of lobular or bronchopneumonia. The inflammation is manifested by the attraction of large numbers of leukocytes which fill the air passages and a swelling and infiltration of the interstitial tissue with serum, leukocytes and red blood-cells. Thus in the early stage of pneumonia, when the hemorrhagic element is predominant, we speak of red hepatization and when later the leukocytes are gathered in large numbers of gray hepatization. As these leukocytes destroy by phagocytosis and ferments the infecting organisms, resolution takes place. There are several factors which retard resolution, among them being a passive congestion due to weakened circulation, extensive thrombosis of the small venules and arterioles, the introduction of new bacteria into the already damaged tissue, especially where this is associated with fatigue or chilling of the patient or insufficient nourishment. The experimental work of Cecil and Blake has shown that during resolution the original bacteria may be entirely replaced by secondary organisms and normal resolution is delayed or thwarted. Frequently resolution ceases and the air channels remain filled with pus. In time this becomes organized with new blood-vessels leading to fibrous changes and the loss of the normal epithelium from the alveolar walls. This leads to the constriction of bronchioles and thus local pocketing of pus, and the lung tissue becomes cirrhotic or fibrous. A heavy tough less aërated portion of the lung thus develops with a chronic infection persisting, and because of failure to drain properly and impaired local circulation, this chronic pneumonia resists the reparative efforts of nature. This type of chronic pneumonia has been very frequently seen following the epidemics of influenzal pneumonia; it generally selects one lobe of the lungs (the lower right most frequently) and clinically is difficult to differentiate from bronchiectasis and localized emphysema. Each of these conditions is necessarily present as evidenced from the pathology.

A so-called primary chronic pneumonia has been described which is not associated with tuberculosis or syphilis. The infecting organisms are possibly less virulent, even at the onset, and the reaction to them is not sufficient to produce the normal processes of resolution. Thus the condition is chronic pneumonia from the onset. Frequently such bacteria, spreading by local extension or by being inhaled into normal tissue, invade the zone about a lung abscess and the fibrous, pus-pocketed areas thus set up greatly hinder the repair of the abscess. This pneumonic zone is very frequent in tuberculosis surrounding either a small or large tubercle or cavity. A tuberculous pneumonia is a very good example of a chronic pneumonia, both as to onset and resolution, resolution resulting not in absorption of cellular elements but in their replacement by fibrosis.

Chronic pneumonic areas not infrequently develop as a result of pleural effusion either in the parietovisceral region or in the interlobar space. Infection in the compressed parenchymatous area may reach the effusion and transform it from a sterile fluid to an empyema. For this reason it is well to remove accumulated fluid and thus avoid the complication of either chronic pneumonia or empyema.

Symptoms and Physical Signs.—Following a history of pneumonia the cough and expectoration continue, the latter becoming purulent and frequently nummular. A low-grade fever, 99.6° F. to 100° F. persists, running a hectic course. The strength is reduced and night sweats may appear from time to time. The sputum may increase to a considerable amount and have a foul odor suggesting lung abscess. It is often noticed that this is more easily raised when gravity is employed—lowering the head so that the mouth is below the level of the lower lobes. Comparative or apparently absolute freedom from symptoms may persist for several months only to be followed by a return of the cough and expectoration accompanied by a moderate fever. This is most apt to occur in the winter and be associated with fatigue, overchilling, or acute upper respiratory infection.

Physical signs are frequently out of all proportion to the symptoms, at times being no more than slight dulness and a few coarse indeterminate râles. Breath sounds are diminished over the area involved, generally at the base, and are of a bronchovesicular type. If a consolidated area communicates with a bronchus a small patch of bronchial breathing is present. Whispered and tactile fremitus are generally diminished because of some thickening of the overlying pleura or because of diminished aëration of the region. There are small areas of relatively normal lung intermingled with the fibrous tissue. Dulness, next to diminished breath-sounds, is the most common sign. The râles are generally coarse and sticky and with a deep breath small atelectatic areas are heard to snap open. From the above physical signs it is often difficult to differentiate thickened pleura either adherent or with a plastic exudate. Also bronchiectasis, associated with, or the result of local emphysema, will frequently give the same signs, the expiration being more prolonged when the latter is present.

Prophylactic Treatment.—Every case of pneumonia should receive careful watching until all signs of moisture have cleared and he should be confined to bed for several days or weeks, if necessary, until a normal temperature is permanently established. Just as during the acute stage of a pneumonia, so during the convalescence the mouth and teeth should receive special care, for the accumulation of food particles or pyorrhea is often the causative agent of chronic pneumonia. Rest is a very important factor in preventing this condition and the patient should not undertake his arduous occupation until he has shown by gradual exercise that he is able to do it without undue fatigue. This sometimes means extra weeks or months but the time is well spent if it will prevent such a complication. When the process of resolution is apparently delayed, although not as yet chronic, there is an advantage in a warm climate where the patient may be out of doors day and night. Good simple food augmented with plenty of milk and eggs is essential in building up resistance and strength. As the reinfection in the lungs frequently is the result of infection in the nose or throat these foci should be investigated and if found, removed at the most opportune time. Lighting-up of a smoldering infection in a chronic pneumonia is often the result of an ether anesthesia and consequently local anesthesia or gas-oxygen should be used by preference.

Active Treatment.—Active treatment for the existing condition is largely symptomatic. Rest in bed is essential while there is fever. Forcing fluids is very helpful to reduce toxemia. Local counterirritation over the affected region by mustard plasters is beneficial for relieving local congestion. During the febrile period leukocytic extract has been used by some with success. The process is rarely extensive and shortly settles down into an afebrile state. Drainage by posture is a very important method to prevent new areas becoming involved and healing is favored by keeping the small cavities and pockets free from pus. The knee-chest position or pronated posture with the hips raised and the head over the side of the bed for fifteen to twenty minutes twice daily will thus prevent much of the constant coughing and expectoration. Autogenous vaccines have been used with success by some as an adjuvant to build up specific resistance. In their preparation a mixed vaccine consisting of the chief bacteria found on blood agar plates is preferable to an isolated type of bacterium.

In very early cases when the condition has been recognized, compression of the lung by artificial pneumothorax may be helpful, but the fibrosis frequently present hinders collapse and favors obstruction to drainage in some cases. Thoracoplasty in very chronic cases with compression of the lower lobe, when alone involved, offers a possible hope of cure. Lobectomy, often followed by thoracoplasty, offers also a possible solution in some of the very fibrous lobes where pus-pockets cannot be drained by pneumotomy.

PULMONARY EMPHYSEMA

Philip H. Pierson

Emphysema, from the Greek "Emphusao" meaning "to inflate," as applied to the lungs refers to a stretching and finally atrophy of the alveolar walls and blood-vessels lying within them. In some instances, senile emphysema, the atrophy may be a part of the general atrophic condition of the tissues and sclerotic blood-vessels predisposing to this distention. Some forms of emphysema are gradual in onset, as in the slowly decompensating heart with attacks of cardiac asthma, senile emphysema, and localized emphysema in chronic bronchitis which results from sinus or other upper respiratory infection. The type of emphysema described by Freund, and discussed more in detail later, is also of gradual onset; the primary pathology here is considered to be in the rib cartilages, which become ossified, distending the rib cage in the state of full inspiration, and expanding the lungs as a consequence of their following the shape of the chest. Mechanical pressure on the trachea, by an enlarged thyroid or mediastinal tumor, or nasal or laryngeal obstruction, may cause a gradually increasing emphysema. A more rapid onset is seen in influenza bronchopneumonia, whooping-cough, laryngeal diphtheria with stenosis, and asthma. The elastic fibers of the bronchioles are generally gradually weakened by toxins, fibrous infiltration or necrosis, and the strain of constant coughing, straining at stool or while lifting heavy weights, or dyspnea, rather suddenly overdistends the bronchioles and alveoli and emphysema results. Practically all emphysema has a superimposed chronic bronchitis, but it is not essential for all chronic bronchitis to be associated with emphysema.

Of the acute forms of emphysema that which was a result of epidemic influenza pneumonia should receive special mention. The experience of all clinicians bears out the fact that this disease varied considerably in different localities, both in the organisms predominating and in the type of pneumonia and complications manifesting themselves. In general it may be said that the pneumonias were largely lobular, that the interstitial tissue showed considerable reaction and that, as a consequence, frequently minute abscesses were scattered through a larger or smaller portion of the lungs. Two types of emphysema have been noted, one which appeared between the second and fourth day and considerably masked the signs of consolidation, and in some instances went on to a very diffuse vesicular emphysema, followed by an interstitial emphysema spreading from the supraclavicular fossæ up to the scalp and down to the pubis, and internally through the retroperitoneal tissues. This type has been described from clinical experience by Torrey and Grosh and experimentally by Kelman. Briefly, it was observed that the infection greatly weakened the interstitial tissue with multiple areas of necrosis and that a rather selective action of the toxin was exerted on the respiratory center causing dyspnea and from its vagal action a slow pulse. This theory was further supported by its appearance early in the disease as opposed to the frequent cardiac dyspnea and cyanosis which occur late in pneumonias. This dyspnea had two effects: one, the production of the emphysema by increased pressure on the weakened tissues, and secondly by its tendency to increase pulmonary blood-pressure there was a constantly increasing passive congestion and cyanosis. This intense emphysema further led to the rupturing of interstitial tissue, favoring the spread of air along the main blood-vessel trunks to the mediastinum and thence up about the subclavian vessels to the supraclavicular fossa and about the abdominal aorta to the retroperitoneal space. When the subcutaneous emphysema appeared the pulmonary pressure was considerably relieved and the oppressive feeling partially disappeared. The pulmonary emphysema quickly abated when the toxicity of the infection was reduced thereby, at least theoretically, substantiating the idea of medullar irritation.

The second type of emphysema related to influenza is the relatively small localized area frequently seen at the base of a lung, previously the site of a pneumonic process. These cases are not uncommon and are the result of the pathology described above. They give rise to exacerbations of infection either chronically located there, or the result of sinus or nasopharyngeal infection. These areas may persist for a number of years and the best method of avoiding exacerbations is to keep the patient in as good health as possible and to remove all foci of infection.

Prophylaxis.—The term "chronic bronchitis" is one that seldom should be used unless qualified by the cause—due to passive congestion, infection in the tonsils, sinuses or teeth, tuberculosis with secondary infection, rarely syphilis, etc. As chronic bronchitis frequently produces an emphysema, attention to these causes is essential. Digitalis by strengthening the heart will relieve considerable of the passive congestion, a potent factor in the infection. Special attention should be called to the sinuses and allied foci as an etiological factor in chronic bronchitis and emphysema. By this it is not implied that all emphysematous patients should receive radical operative procedures without a thorough examination, but in many it is a causative agent. After the focus has been cleared up autogenous vaccines may be of some help in relieving the chronic infection. This is also true in tuberculous patients. The emphysema seen in this group of patients is largely a compensatory aid on the part of nature to make up for the amount of tissue disease. Sometimes it is in small areas in one lung almost enveloping the tuberculous process, or it may be largely manifested in the better lung where there is considerable destruction on the opposite side. Where pleural effusion, or artificial or spontaneous pneumothorax compresses one lung, the opposite side shows a moderate or extreme compensatory emphysema depending upon the rapidity of the collapse. As mentioned above, transient emphysema is frequently found in pneumonia. In discussing emphysema in infants, Alexander and Follet have attributed it to a rupture of some of the pulmonary vesicles, this being due to a congenital weakness of the lung parenchyma. Whooping-cough is a frequent cause of this condition in children. If any of these transient types of emphysema exists for some time, it becomes chronic by the relaxation and wasting of the elastic fibers in the interstitial tissue and the atrophy of the alveolar walls.

Freund's Theory and Operation.—In 1859, Freund advanced the idea that the primary cause of many cases of emphysema was a gradual ossification and elongation of the costal cartilages with a fixation of the chest in inspiration. For many years his theory attracted but little attention, but, since 1900, others, including Garré, Fraenkel and Korte, have carried out the idea with some success. Freund felt that in young adults, even as early as sixteen to eighteen years of age, a change took place in the rib cartilages, beginning with a dirty yellow degeneration which progressed to ossification either complete or interspersed with vacuoles, and that with this calcification there was an elongation and twisting of the cartilage, pushing the sternum forward and to one side, if the elongation was more on one side than the other. He found that the right side was often the more involved. As inspiration required more muscular effort, and as dyspnea and air-hunger began to develop in these individuals, this part of respiration received more attention and an increase in the residual air developed. With this increase and the gradually enlarged and rigid thorax, expiration, generally a passive motion and dependent on lung elasticity, became more and more difficult and the resulting emphysema was consequently secondary to the diseased costal cartilages. This led him to devise an operation whereby pieces of cartilage were resected, generally on the right side from the second to the fourth or sixth rib, thereby allowing the thorax to take the position of mid-inspiration, and again favoring freer expansion and contraction of the lungs. Before operation a careful survey of the thorax should be made to determine (1) that it is rigid; this is determined by palpation, inspection, mensuration and fluoroscopy; it is considered immobile if the expansion at the level of the fourth rib is not more than 1 cm. Fluoroscopically, almost no motion is seen laterally and the diaphragm becomes flattened and pushed down and very little movement is noticed; (2) that the cartilage is ossified; this is done by palpation, X-ray pictures, "L'akidopeirastigue" (puncture of the cartilage by a special needle determining by the resistance encountered the amount of calcification present); (3) that the thorax is distended; this is observed by an increase in the angle between the ribs and sternum both at the upper and lower end, and the consequent more horizontal position of the ribs. The condition of the heart is an important factor determining operation. Weakness of the heart muscle develops partially because with lack of normal lung elasticity and breathing there is a diminished suction of the blood to the heart and also a lessened flow through the lungs. The congestion manifests itself peripherally and by a dilatation of the superficial venules about the site of attachment of the diaphragm, frequently called "athletic belt." Also the vital capacity has been investigated, and whereas a normal estimation averages 3,500 to 4,000 c.c., with 500 c.c. less in the reclining posture, where there is slight rigidity this is reduced to 2,400 c.c. and even to 800 c.c. with marked rigidity.

Patients with definite myocardial changes and purulent bronchitis are not considered good risks. The operation has been performed under general as well as local anesthesia. Several deaths were reported as due to the immediate effects of the anesthetic when ether was used, and so local anesthesia is deemed safer. For a full account of the technic of this operation the reader is referred to an excellent book by Sauerbruch. The end results after two to three years show a considerable proportion of cases so much relieved that they may return to moderate work and usefulness. Union at times takes place between the ribs and sternum, reimmobilizing the thorax, but even then the extremely distended position is not assumed and the discomfort is not so great. After treatment in the form of breathing exercises, etc., is very important.

Relatively little of this work has been done in this country, and, while cases should be carefully chosen and studied before operation, it may offer more relief in many cases than can be obtained by medical means.

Treatment.—In addition to the above suggestions, occupation, hygiene and climate should be mentioned. Dusty occupations, those requiring heavy lifting or straining, where there is exposure to extremes of heat or cold, are to be avoided. It has been said that glass blowers and musicians using wind instruments are more prone to emphysema, but more recent practical observations have failed to verify what theoretically seems very probable. Where the climate prevents considerable out-of-door life, or offers marked changes from heat to cold, or a great deal of rain, a more equable environment should be sought. Certain asthmatics are seasonably troubled by the pollens of certain plants or flowers. Some of this hypersensitiveness may be overcome by desensitizing vaccinations. If it still persists a change to a locality free from such irritations is advisable. Permanent residence in Southern California or frequent trips to Florida during the winter are helpful in many instances. As a further aid in preventing acute respiratory infections, cold baths in the morning are beneficial. In some it is necessary to begin with cool water and gradually approach the cold. A good reaction is favored by standing in warm water and dashing the cool or cold water over the body and having the room fairly warm. Abdominal distention, either by gas, ascitis or large tumors should be corrected because by their pressure the diaphragm becomes less able to function and the ribs are pushed up and out thereby favoring emphysema.

Mechanical Devices.—Numerous forms of apparatus have been devised and exercises suggested, the object of all of them being to retard inspiration, favor expiration and thus increase pulmonary and peripheral circulation.

Rossbach has devised a chair with a high back in which are placed two vertical rounds. These are connected both with levers which the patient himself can operate and with a swathe which compresses the chest. By pushing forward the levers, the chest is compressed and by relaxation inspiration is favored. Its simplicity and ease of operation justify its trial in some cases. Perhaps of more value is the apparatus of Boghean which is automatically driven, having the double advantage of establishing a rhythmical contraction and relieving the patient of the effort to operate any levers. Swedish gymnastic exercises have been helpful, per se, and in conjunction with mechanical devices. By these the patient or his masseur rhythmically exerts pressure on the anterolateral aspects of the thorax favoring expiration. Following an acute asthmatic attack it is helpful to use this pressure in relieving the lungs of the increased residual air which has resulted during the seizure. In this way there is less constant pressure in the alveoli between attacks. By massage the peripheral circulation is improved thus indirectly favoring the pulmonary circulation. Hoffman suggested exercises with dumb-bells weighing about one pound. While bringing the arms to a horizontal position, with the palms up, the patient slowly inspires. Expiration is accompanied by bringing the arms to the side, bending the knees, and pushing the dumb-bells slowly down till they touch the floor. All these exercises, whether or not aided by a machine, should be done slowly and should begin with only a few each day and gradually increase their number. Kingscote suggested a series of breathing exercises to help open numerous alveoli which he felt were compressed by the dilated ones. While in the upright position, preferably, a towel is placed about the lower ribs, thus forming a band about six to eight inches wide. While the patient expires the towel is tightened and fixed. Then with inspiration, if the thorax is not rigid, the upper part must expand and, according to his theory, open those alveoli which have been idle for some time. Their expansion will close numerous dilated ones and by coughing much of this débris will be expelled. Hence to avoid further inspiration of this pus and débris, inhalations should be slow and only a few, at intervals, daily. In his hands, over a period of twelve years, and used in over 1,000 cases 91 per cent have been "restored to health." It may be used with caution in chronic fibroid tuberculosis and also in persons of sedentary habits who use only a relatively small part of their lungs for ordinary breathing.

Gerhardt, Romanoff, Cloetta, et al. have suggested that as the diminished pulmonary circulation is an important factor in the production of emphysema, an amelioration of the condition may be effected by improving the blood supply. Claude Bernard has produced emphysema in animals by partial ligation of the trachea and Cervello has produced the same condition in dogs by inducing nasal obstruction. Consequently, Gerhardt advised the lessening of intratracheal pressure and found that the pulmonary circulation was increased. Several forms of apparatus have been devised to aid this procedure. A pneumatic cabinet, by Waldenburg, in which the patient sits, is so equipped that the air pressure may be gradually raised to one and one-half to two pressures during one-half to one hour, and he breathes at that pressure for one to two hours and then the pressure is gradually lowered to normal again. In another device, he inspires at a positive pressure of about one-sixtieth to one-fiftieth of an atmosphere and exhales into a negative pressure of about one-fortieth to one-fiftieth of an atmosphere. In a mouth-piece described by Bruns, the patient expires against a partial vacuum, this being very similar to that suggested by Kuhn, in which inspiration is partially obstructed but expiration is unhindered. The latter exerts only periodic dilatation of the blood-vessels. The device which has attracted most favorable comment is one of Hofbauer's. In this "expirator" there is a light and bell which accompany expiration, thereby aiding the patient to produce this act in a slow, even manner, rhythmically. He is also instructed to hum during expiration, this favoring nasal breathing. In order to facilitate diaphragmatic breathing, a small pillow, pressed against the abdomen, is inflated with each expiration until the patient can combine these forces in a more normal and rhythmical expiration. Von den Velden suggests putting partial tourniquets about the limbs during cardiac asthma, thus reducing the tendency to marked passive congestion, itself a predisposer to emphysema.

Drugs.—For the cardiac weakness digitalis is useful both in preventing and relieving the emphysema associated with it. Atropin by its paralyzing action on the vagus and enlarging the bronchioles is very helpful. It also dries up the moisture which is present in certain types of emphysema. Adrenalin is helpful in the same way by stimulation of the sympathetic ganglia. Camphor paralyzes vasomotor nerves and enlarges the blood-vessels and so has been suggested by Liebmann. According to Webber, Cloetta and Anderes, the lungs possess vasomotor nerves. Potassium iodid in small doses (gr. x three times daily) is helpful in loosening secretions, and thus aiding expectoration. It also tends to relax bronchiole spasm and thus facilitate breathing. The sputum in emphysema is very tenacious and ropy when acute attacks of asthma are superimposed, and straining to expectorate should be avoided. Ammonium chlorid and carbonate in gr. v doses and fluid extract of grindelia in half dram doses are helpful expectorants. For the accompanying bronchitis and bronchiectasis the reader is referred to these subjects. Maintenance of general nutrition and hygiene with special reference to mouth cleanliness is an important factor.

GANGRENE AND LUNG ABSCESS

Philip H. Pierson

Clinically, lung abscess and gangrene have been considered together by most observers, their chief difference being one of pathology. The former may be implanted on a normal lung, but the latter is favored by trauma of the tissues or the original implantation of bacteria more specifically associated with gangrene. While abscesses may be multiple, they are more frequently discrete, without so much destruction of large areas of tissue, en masse, as occurs in gangrene.

Gangrene

Factors predisposing to gangrene are the very much undernourished general condition of the patient, metabolic diseases, and the aspiration of certain substances and types of bacteria. Diabetes and alcoholism, according to Sauerbruch, not infrequently lead to *Pneumomalazie,* or small areas of softening which later become gangrenous. Whether the abscesses seen at times during nephritis are due to lowered resistance or toxic blood-vessel changes, possibly thrombosis, is not definitely established. A healthy lung can fight gangrene even though it be infected with pus. Where the lung has been traumatized by crushing injury or bits of shrapnel or bullets, carrying in pieces of bone or flesh, and thus interfering with the blood supply, gangrene is more to be anticipated. Kissling states that saliva from pipe smokers, inhaled during sleep, may produce gangrene. The thin serosanguineous discharge from a pharyngeal ulceration may be inhaled and lead to this condition. The bacteria found in gangrenous lung tissue include flora similar to that found in the tartar of teeth, spirochetes, comma bacilli, fusiform bacilli and yeast molds. These bacteria have a quite distinctive odor and may frequently be recognized on the breath or from the sputum.

Lung Abscess

The etiological factors in lung abscess can in general be grouped under three heads: aspiration, pneumonia or embolism. While a secondary pneumonia always exists about both aspirated material and emboli, the type of pneumonia referred to is generally bronchopneumonia (lobular), such as existed during the influenza epidemic, caused frequently by the streptococcus with or without the aid of Pfeiffer's bacillus or a Friedlander bacillus, but seldom caused by the pneumococcus. The pathology of these influenzal abscesses was carefully described by MacCallum.

Aspirated material may be blood, mucus or infected cheesy material from tonsils during their removal under a general anesthetic and unconsciously inhaled where the laryngeal reflexes are abolished; foreign bodies such as teeth or purulent discharge during extraction, buttons, marbles, tacks, kernels of corn, peanut shell, etc., and particles of food aspirated into the lungs during laughter or from the mouth during sleep. As thus implied, sleep, alcoholic and epileptic unconsciousness, as well as that from anesthetics, and such diseases as hysteria, certain brain tumors and diphtheria, with pharyngeal paralysis, are definitely predisposing factors in aspiration lung abscess. While material may be inhaled during tonsillectomy under a local anesthetic, this is less likely, especially where an efficient suction apparatus is constantly operating. Coakley (quoted by Hedblom) and others are more inclined to believe that these posttonsillectomy lung abscesses are due to aspiration than emboli. It is possible that a very carefully given anesthetic which does not abolish all laryngeal reflexes (Botsford) and a recumbent position may partially overcome this danger in children and others where local anesthesia cannot be used. The inhalation of foreign bodies is a very important and often overlooked cause of lung abscess and should always be kept in mind where signs of lung abscess or chronic localized bronchitis or bronchiectasis have existed for a long time. Hedblom reporting a series of 40 cases of lung abscess, at the Mayo Clinic, due to foreign bodies states that 23 cast no shadow by the X-ray and that, in 14 of these 40, it had been present for at least seven months and in 7 more cases for more than two years. Bronchoscopy is therefore indicated in some instances where the X-ray is negative but where there is a reasonable suspicion of foreign body. Aspiration may also take place from a neighboring bronchiectatic cavity, or from one abscessed area into a portion of normal lung, there setting up another focus. Lung abscess may result from the breaking into the lung of an empyema, either interlobar or parietal; by the extension of a phlegmon of the chest wall, or the perforation of subdiaphragmatic abscess, peptic ulcer, appendix abscess, or other pus infection in this region; or by the erosion of the esophagus and trachea by malignancy or retropharyngeal abscess. I have recently seen a patient in whom lung abscess was clinically an aspiration posttonsillectomy condition, but pathologically was apparently implanted on an old healed tuberculous cavity which had given no signs or symptoms for at least four or five years. Without the pathologist's report this complication would not have been suspected from the clinical evidence.

Emboli from fresh endocarditis lesions, mural thrombi, otitis media or thrombophlebitis of the extremities, pelvis, scalp or jugular vessels may produce lung abscess from the combined disturbance of blood supply and bacteria introduced. The areas involved as infarcts are cone-shaped with the base toward or on the pleura, and therefore, if the displaced thrombus is of some size, physical signs may be more definite than in aspiration. As mentioned under Thrombosis and Embolism postpartum emboli as a cause of lung abscess may be one of the first signs of a beginning pelvic phlebitis. These patients present a graver outlook than with foreign bodies because of the complication with the predisposing cause. Echinococcus cysts have been transplanted to the lung by the blood-stream and their growth may very much simulate an abscess. Finding the organisms in the sputum is an important aid in differentiating this condition from an abscess.

Location.—In general, circumscribed abscesses are seen more frequently in strong individuals with good resistance, and diffuse, progressive, spreading abscesses without clean-cut borders in people with poor general reactive powers. Wessler's figures as to the tendency of aspiration and non-aspiration (including embolic and pneumonic) abscesses to be located in the upper or lower lobes is fairly consistent with other observers. In 101 cases his observations were:

LOCATION OF ABSCESSES

Abscesses	Upper Lobe	Lower Lobe	Middle Lobe
Aspiration	18	9	3
Non-aspiration	24	44	3

Naturally, larger emboli and larger aspirated substances seek the larger vessels and the most accessible branches of the bronchi. The right lung is the site of more than half of the aspiration abscesses. Recently, the author observed a case of abscess in the right lower lobe from the aspiration of polluted water which was inhaled when the patient fell from the gangplank of a ship.

Symptoms and Physical Signs.—Acute symptoms of lung abscess or gangrene may not appear till the thirteenth or fourteenth day after the operation or pneumonia, and this may in part be due to the slowness of growth of some of the anaërobic bacteria present. The onset is generally gradual, with fever, increased pulse rate, cough and at first mucoid sputum. A foul odor may appear on the breath slightly before the pus begins to drain. Such a history, followed by the sudden coughing up of a large quantity of pus (8 to 10 ounces at times), is very typical and almost pathognomonic of lung abscess. With the coughing up of the sequestra the temperature may fall to normal or be reduced from 103° to 104° F. to 99° F. to 100° F. Frequently, night-sweats appear early in the disease. The individual with poor resistance may not react so abruptly to the infection. Hemoptysis of a teaspoonful or more is not uncommon, and by some authors has been said to be present more often than in tuberculosis. The reason for this may be that, in the latter disease, its chronicity tends to obliterate the blood-vessels in the walls, or those passing through a cavity more than is the case in acute infectious lung abscess. The foulness of the sputum, which is not alone very unpleasant to the patient but to his attendants, together with the toxicity, soon produce anorexia, and considerable loss of weight and strength may develop. This is particularly true of gangrene and its presence should make one suspect this condition. Aching in the joints at times with swelling is a frequent symptom of the toxemia.

According to Sauerbruch, the particular symptoms of gangrene to be added to those of abscess are: (1) the process is more gradual as compared with the pneumonic onset of abscess; (2) the loss of weight and strength is much more rapid, a well person being changed to a very sick one in three to four weeks; (3) the heart is early affected and with the dyspnea produces considerable cyanosis; (4) headaches and foul perspiration are common; (5) the amount of sputum rarely is more than 250 c.c. daily.

On physical examination one of the most important signs, if present, is circumscribed pain on pressure over the abscess. This is emphasized by Sauerbruch and is in accord with the work of Pottenger, bearing out the presence of sensory as well as trophic and motor reflexes from the lungs. The most common sign is dulness and if the abscess is centrally located it may be the only sign. If nearer the periphery of the lung there may be diminished bronchial breathing which is more apt to be heard if the abscess is of embolic origin. Pleuritic crepitations (evidence of a plastic pleurisy) may be heard over the suspected area. Whispered fremitus will be increased before the type of breathing is changed. An important physical sign is the appearance of amphoric breathing or whisper, or the change from dulness to resonance following cough, or the expectoration of considerable pus. This is not common. Clubbed fingers appear along with the occasional periosteal thickening of long bones as early as six weeks in some cases. In one of my cases they appeared in two weeks.

Of the laboratory aids, X-ray has helped considerably in the localization of abscesses. When the patient is lying down films should be taken with the rays traveling horizontally (to obtain fluid level), and in this way both lateral and posterior-anterior views will aid in determining the position of the cavity. Stereoscopic views are preferable to flat plates. In the fluoroscope localization is also aided by rotation of the thorax and by noticing the cavity "wink" if it communicates with a bronchus. As the cavity may be filled at the time of the first observation, a second examination is always necessary before it can be excluded.

Rapid destruction of tissue will generally provide elastic fibers in the sputum but where the process is gradual they may be absorbed. By placing the sputum in a glass measure there are generally three fairly sharply defined layers seen: the upper consists of foam, the middle layer is of pus and the lower of mucus. This is not typical but frequently happens. The sputum from abscesses of the upper lobes is generally more constant and in small amounts, while from the lower lobes there are periods of relative freedom and then large amounts, particularly if drainage posture is used. Leukocytosis may or may not be present, depending somewhat on the reaction of the patient. The urine contains albumin and the spleen is generally enlarged. It is safer not to attempt exploratory puncture because of the danger of hemorrhage and the infection of the healthy lung or pleura.

Differential diagnosis of lung abscess and ruptured empyema is frequently difficult. The onset and localization of the fluid level may be of some help. Ecchinococcus cysts may have less of an inflammatory reaction about them, and the disease may be found elsewhere, liver or intestines, and the organisms may be found in the sputum. Bronchiectatic cavities may turn into abscesses but generally lack elastic tissue fibers. (For differentiation from chronic pneumonia, see preceding section.) A fetid bronchitis may turn into gangrene. Hedblom found three tumors in his series of 40 operations, primary and secondary carcinoma and a primary sarcoma. These must also be kept in mind in the diagnosis.

Medical Treatment.—Removal of the sequestrum and adequate drainage are the two essential factors in the treatment of lung abscess or gangrene. Following this the aim is to approximate the walls of the abscess. This, nature may do herself, or artificial means may be necessary. The removal of the sequestrum is often accomplished spontaneously by coughing it up and, in general, unless there is a great deal of toxemia and rapid spreading of the process it is advisable to be conservative. It is permissible to delay operating for four to eight weeks if everything is progressing favorably, that is, steady diminution in sputum and especially in the pus, normal temperature, and improved general well-being. During this period of waiting, the treatment is very similar to that of pneumonia; plenty of fluids and liquid or light foods with high caloric value, expectorants if necessary when the sputum is at all tenacious, rest at night, if necessary, with sedatives (codein, gr. 1/4 to 1/2, or morphin, gr. 1/8 to 1/4). Some use creosote, either as the carbonate 5 to 15 drops on sugar or as the suspension "creosota pura" in olive oil, 7 minims hypodermically daily. Myrtol, at present difficult to procure, in doses of 2 minims, two or three times a day is a very efficient deodorizer for the sputum. A small steaming kettle with eucalyptus oil, or benzoin in the water, or a cresolene lamp is pleasing to the patient and an effective means of disguising the putrid odor. Medicated masks are to be avoided because they are therapeutically of little or no value and because they diminish the amount of fresh air obtained. Posture is a very important part of the medical treatment. In lower lobe abscesses it is wise to raise the foot of the bed, beginning with two inches and increasing to six inches for from fifteen to sixty minutes twice daily. The patient should lie on the side of the unaffected lung. Strapping the affected side will frequently relieve the pleuritic pain and further help nature splint the lung and check the spread of the disease by aspiration into new areas. The after-care for those treated medically is the same as for surgical cases. A further aid in immobilizing the diseased lung is artificial pneumothorax. Before the wall of the cavity has become too firm, and provided adhesions do not prevent collapse, considerable aid may be obtained by its use. Forlanini's famous case that was cured after six years of expectant treatment has served as an encouragement for further using this compression. The chief difficulty encountered is the adherent pleura over the abscess which practically always exists. This may not be a complete contraindication for in many instances this is a local adhesion and air may go above and below it. This inability to compress the lung but partially may result in an obstruction to the drainage and lead to further pocketing off the infection. A spontaneous pneumothorax may result from the tearing loose of a small adhesion and with this an empyema. This lends a very grave aspect to the prognosis.

Surgical Treatment.—If this period of medical treatment is not efficient in obtaining the desired results, or if at the start it is contraindicated by the presence of a foreign body or a rapidly progressing lesion, surgery is indicated. If possible it is advisable to wait until the abscess is walled off, a condition best judged by the X-ray. The anesthetic of preference is local, although nitrous oxid and oxygen or ether may be used, as it is at times desirable to have the patient's coöperation, either by coughing or breathing against resistance. Whenever possible the operation should be done in two stages, the first to assure sufficient pleural adhesions to prevent infecting the pleura and the second to open and drain the cavity. Sauerbruch uses the differential pressure apparatus, and after opening the parietal pleura packs the field with gauze rather than sewing the two layers together. This he feels is a safer method in preventing pleural infection. Many other surgeons do not use the differential pressure but sew the two pleural layers. The drainage tract is generally opened by the cautery. The site of operation in upper lobe abscesses is generally either in the back or axilla, while practically all lower lobe abscesses are opened posteriorly. If drainage is sufficient, signs of improvement may be expected very early in convalescence, especially a diminution in the amount and quantity of pus in the sputum. A subsequent rise in temperature may be due to too tight packing of the wound or to a new focus. As many as four separate abscesses have been operated upon in the same patient with recovery. Hemoptysis may be controlled by pressure in the wound or by the same means as when a symptom of pulmonary tuberculosis. In favorable cases the wound may be closed in six to eight weeks. In thick-walled cavities, which show very little or no signs of closure, the whole cavity may be excised, and the wound closed with a skin and muscle flap. In other cases thoracoplasty is advisable to collapse the cavity. For the more recent technic in this operation as well as that for lung abscess and gangrene, the reader is referred to Sauerbruch, Korte, Fraenkel, Lenhartz, Robinson or Hedblom.

Results of Treatment.—With medical treatment the mortality is as high as 60 per cent to 90 per cent with in general only about 10 per cent spontaneous cures. The recent figures of Garré and Sauerbruch where surgery was used are:

MORTALITY

Author	Character of Cases	Number of Cases	Cases Cured	Cases Died	Per cent Mortality
Garré	Abscesses	122	80	42	34
Collected up to 1919	Abscesses	182	132	50	17.5
Sauerbruch	Gangrene	281	197	84	29.3
Collected April, 1919, to 1921	Abscesses, acute	19	16	3	15
	Abscesses, chronic	53	30 3 with bronchial fistulæ, 2 in good health	20	37
	Gangrene	37	18	19	51

The glycosuria which accompanies some lung abscesses, whether diabetic or not, frequently clears up without dietary changes after the cure of the lung abscess.

Postoperative Treatment.—This is very important for upon it depends much of the success of the operation. It is advisable to have a specially trained nurse who will constantly urge the patients to expectorate as much as possible. Enough morphin should be given to relieve pain and thereby allow the patient to cough. Simple food at frequent intervals and of high caloric value is very important. Tonics of iron, quinin and strychnin, or arsenic are helpful. Living out-of-doors, provided the weather is not too severe, and later spending several months in an equable climate is very advisable. Return to work or ordinary activities must be delayed for some time, for with a lowered general resistance a smoldering focus may light up and spread. While it is possible for many patients to become absolutely cured, there is still much to be desired in improving the treatment of lung abscess and gangrene.

TUMORS OF THE LUNG

George Blumer

Hitherto the treatment of lung tumors, whether malignant or non-malignant, has been, with a few exceptions, palliative. This was necessarily so for two reasons: one, the great difficulty of making an early diagnosis; the other, the imperfect development of the methods of lung surgery.

The intensive work done during the war has thrown much light both on certain methods of diagnosis and also upon the surgery of the lung. The development of X-ray technic has been rapid, and it is now possible not only to decide whether a pulmonary neoplasm is present, but also to localize it pretty accurately in most instances. The chances of early diagnosis have, therefore, increased greatly.

The surgical experiences of the war have demonstrated that differential pressure is not so essential as was formerly believed; that the pleura is well able to care for infection and that operative pneumothorax is not nearly so serious as it was formerly thought to be. Nevertheless conservative surgeons, Matas for example, insist that there is still a place in civil surgery for the use of differential pressure. The use of differential air pressure was first placed on a firm basis by Sauerbruch in 1904. Its object is to remove what has hitherto been the greatest obstacle to successful intrathoracic surgery, namely, the traumatic pneumothorax which results from opening the chest. This can be prevented, as Sauerbruch shows, in one of two ways: either by positive air pressure within the air passages, or by negative pressure outside of them. As to the relative value of the two kinds of pressure there is still room for discussion, and some observers, Willy Meyer, for example, are convinced that the two forms of pressure do not act exactly alike in human beings. It is sufficient for practical purposes to know that both forms have been successfully used in thoracic surgery.

So far as portability and cost of apparatus are concerned, the positive pressure mechanism in its various forms has a great advantage over the negative pressure apparatus. In principle the positive pressure apparatus consists of a means of introducing the compressed air, a compressor with a manometer for registering the degree of air pressure, a duct for the return air protected by a valve, and an anesthetizing chamber usually provided with a by-pass so that the anesthetic can be excluded at will. It would be out of place here to go into great detail regarding the different forms of pressure apparatus.[1] The larger ones take the form of an air-tight cabinet, which contains not only the patient's head, but also the anesthetist. The apparatus of Robinson, which costs about $300, is of this type, and Brauer's apparatus, while it does not contain the anesthetist, is constructed on the same general principle. In the smaller instruments, such as Elsberg's modification of the Meltzer-Auer apparatus, Robinson's portable machine, or Flint's apparatus, compressed air is introduced through a mask or through a cannula inserted into the patient's air passages. War experience has demonstrated the value of these smaller appliances.

The negative pressure apparatus is much more cumbersome and much more expensive than the positive pressure apparatus, inasmuch as it is in the form of a cabinet large enough to contain the body of the patient (his head being outside), and the operator with his assistants and armamentarium. The air is exhausted from the chamber by means of a pump to a degree necessary to overcome the elasticity of the lungs, so that they do not collapse when the chest is opened. Fresh air is supplied through a water valve.

Willy Meyer, of New York, has devised an apparatus consisting of a positive pressure cabinet within a negative pressure cabinet, which allows of the use of either positive or negative pressure at will, or of a combination of the two. The change from one form of pressure to another can be rapidly made without harm to the patient. The Meyer apparatus is more spacious than those hitherto devised. The operator can see the patient's head and face, and the whole apparatus can be knocked down and transported from one place to another.

While there is little doubt that either positive or negative pressure can be used in the performance of an intrathoracic operation, objections have been made to certain features in the different types of positive pressure apparatus, which time may show are of practical import. Brauer's apparatus has been criticized because of the imperfect control which the anesthetizer has over the patient. The Meltzer-Auer method has been criticized because it necessitates the introduction of a cannula into the air passages, a procedure which is at times practically impossible, and which, when possible, may sometimes result in a dangerous hypersecretion of mucus within the trachea.

---

[1] Details of the different forms of apparatuses will be found in the articles mentioned in the references.

The methods employing masks have been criticized on the ground that these may interfere with the proper handling of the patient during vomiting. All of these criticisms are at present academic. Operations on human beings have been done by the methods criticized and without the predicted ill results, but these have been too few in number to discredit the validity of the criticisms, and the question must be left open for the present. It is certainly justifiable to use the simpler positive pressure machine in emergencies where the larger cabinets are not available, and in hospitals where the expense precludes the use of the more elaborate apparatus. The criticism that the positive pressure is not available for extensive pneumectomy on account of hindrance to the collapse of the chest on the operated side has been removed by Flint, who has shown that it is an easy matter to use aspiration and leave negative pressure within the chest in place of the positive pressure.

One other difficulty to successful removal of the lung, the treatment of the stump so as to leave it permanently air-tight, has been overcome by the work of Willy Meyer. He has shown that by means of crushing and invagination perfect healing with an air-tight stump can be secured.

So far as past results in the removal of lung tumors are concerned, there are but few successes to record. Lenhartz successfully removed a carcinoma involving the right upper lobe. The patient survived the operation at least two years. Garré removed a carcinoma of the pleura successfully. Pean has successfully removed a chondroma, and Tuffier an angioma, of the lung. Kuttner has done palliative operations in carcinomata with Brauer's positive pressure apparatus. Future progress in the successful removal of lung tumors depends upon the development of more skilled operators, early recourse to the X-ray in doubtful thoracic cases, and the more widespread development of intrathoracic surgery. Grünmach, Holzknecht, and Otten have all diagnosed lung tumors with the X-ray. There is little doubt that the use of differential pressure will become more widespread, and in time well-trained thoracic surgeons will be found in all medical centers.

Even under the most favorable conditions only a certain percentage of lung tumors are operable. Seydel, who has studied this question on autopsy material, shows that 26.9 per cent of lung sarcomata are operable, and only a little larger percentage of pleural sarcomata. Carcinoma of the lung presents a very much less favorable opportunity—only 9 per cent being operable. Lung carcinoma metastasizes early, is frequently situated at the root of the lung, where it is not very accessible, and is often surrounded by dense adhesions. Carcinoma of the pleura is likewise operable in only about 9 per cent of the cases. However, as Seydel's figures are based on postmortem observations, it is fair to assume that, when our methods of early diagnosis are more perfect, the percentage of operable cases will be considerably higher. Tumors within the bronchi may be sometimes removed through the bronchoscope as Yankauer has demonstrated.

REFERENCES

Circulatory Disturbances

Philip H. Pierson

Auer, John, and Gates, Frederick L. Adrenalin Pulmonary Edema, Journ. Exper. Med., xxvi, 201, 1917.

Branson, G. J. Auricular Fibrillation and Pulmonary Edema, Lancet, ii, 1078, 1919.

Cumston, C. G. Treatment of Acute Pulmonary Edema, Therap. Gaz., xlii, 537, 1918.

Davidson, W. Cameron. Acute Edema of the Lungs, Brit. Med. Journ., ii, 163, 1920.

Drinker, C. K., Peabody, F. W., and Blumgart, H. L. The Effect of Pulmonary Congestion on the Ventilation of the Lungs, Journ. Exper. Med., xxxv, 77-95, 1922.

Fuhner, H., and Starling, E. H., Journ. Physiol., xlvii, 301, 1913-1914.

Giegel, von Richard. Das Lungenodem, München. med. Wchnschr., lxviii, 1078, 1921.

Lord, Frederick T. Diseases of the Bronchi, Lungs and Pleura, 328, Lee & Febiger, Philadelphia, 1915.

Musgrave, Cecil. Acute Edema of Lungs, Brit. Med. Journ., i, 468, 1920.

Powell, Sir Richard Douglas. Advances in Knowledge Regarding the Circulation and Attributes of the Blood since Harvey's Time, Lancet, xi, 979, 1914.

Southwood, A. R. Superacute Pulmonary Edema, Med. Journ. Australia, ii, 284, 353, 1919.

Tribe, E. M. Journ. Physiol., xlviii, 154, 1914.

Weber, E. Arch. Physiol., 1910, Suppl. 410, 383, 1912; 535, 1914.

Wiggers, Carl J. Normal and Pathological Physiology of the Circulation, Nelson Loose-Leaf Living Med., iv, 254, 1920. Thos. Nelson & Sons, New York.

Wilson, D. W., and Goldschmidt, S. Influence of Oxygen Administration—Lung Edema, Am. Journ. Physiol., l, 157, 1919-1920.

BRONCHOPNEUMONIA

PHILIP H. PIERSON

Barach, A. L., and Woodwell, M. N. Oxygen Therapy, Arch. Int. Med., xxvii, 367, 1921.

Bernton, Harry S. Recent Classification and Treatment of Pneumonia, Fiske Fund Prize Essay No. lviii. Snow and Farnham Co., Providence, R. I., 1919.

Boggs, Russell H. Complications of Influenza from Roentgenological Standpoint, Am. Journ. Roent., vi, 239, 1919.

Brooks, H. The Treatment of Pneumonia, Med. Clin. North America, v, 993, 1922.

——— Convalescence in Lobar Pneumonia, Ibid., iv, 1423, 1920-1921.

Burnside, L. A. Note on Value of Prophylactic Vaccination Against Pneumonia, Mil. Surgeon, xlvii, 589, 1920.

Cole, Rufus. Antipneumonococcus Serum, Journ. Am. Med. Ass., lxxvi, 111, 1921.

Duckwall, B. F. Protective Value of Pneumococcus Vaccine Against Respiratory Disease, Mil. Surgeon, xlvii, 589, 1902.

Dudley, Sheldon F. The Biology of Epidemic Influenza, War section. Roy. Soc. Med., Part III, xiv, 37, 1921.

Gillett, H. T. Vaccine Treatment of the 1918 Epidemic of Pneumonia, Lancet, ii, 945, 1920.

Goldsborough, B. W. Treatment of Pneumonia with Antipneumococci Sera, Med. Council, xxv, 805, 1920.

Haldane, J. S. Recent Developments in the Therapeutic Use of Oxygen. Contributions to Medical and Biological Research, i, 549, 1919.

Honei, J. A. Influenza and Bronchopneumonia, Am. Journ. Roent., vi, 226, 1919.

John, Henry L. Glucose as an Adjunct Measure in the Therapy of Pneumonia, Am. Journ. Med. Sc., clx, 542, 1920.

MacCallum, W. G. Pathology of Pneumonia in U. S. Army Camps, 1917-1918. Monograph No. 10, Rockefeller Inst., 1919.

Meakins, J. C. Therapeutic Value of Oxygen in Pulmonary Lesions, Brit. Med. Journ., i, 324, 1920.

——— Shallow Breathing in Pneumonia. Arch. Int. Med., xxxv, 1, 1920.

Opie, E. L., Blake, F. G., Small, J. C., and Rivers, T. M. Chronic Pneumonia, Epidemic Respiratory Disease, 273, C. V. Mosby Co., St. Louis, 1921.

Selby, John Hunter. Hemorrhagic Pneumonitis, Am. Journ. Roent., vi, 211, 1919.

Stadie, W. C. Construction of Oxygen Chamber for Treatment of Pneumonia, Journ. Exper. Med., xxxv, 323, 1922.

Emphysema

Philip H. Pierson

Alexander, M. E., and Follet, E. C. Subcutaneous Emphysema with the Report of Several Cases, Particularly One with Very Extensive Generalized Emphysema, Journ. Am. Med. Ass., lxxii, 930, 1919.

Baumann, A. Un nouveau traitement de l'emphysème pulmonaire, Bull. de Thérap., clxviii, 15, 102, 1914.

Berkley, H. K., and Coffen, T. H. Generalized Interstitial Emphysema and Spontaneous Pneumothorax as Complications of Bronchopneumonia, Journ. Am. Med. Ass., lxxii, 535, 1919.

Braun. Beiträge zur Chondrektomie nach Freund, Deutsche med. Wchnschr., No. 32, 1912.

Creyx. Traitement de l'emphysème pulmonaire, généralisée, Journ. de méd. de Bordeaux, L-50, 351, 1921.

Florand, Antoine, et Flurin, Henri. Rhinobronchites descendantes et Emphysème pulmonaire, Bull. et mém. Soc. med. d. hôp. de Par., xv, 729, 1922.

Gerhardt, D. Pathologie, Pathogenese und Therapie des Lungenemphysems, Ztschr. f. ärzt. Fortbild., Jena, xix, 7, 1922.

Hofbauer, L. Moderne Emphysembehandlung, Wiener klin. Wchnschr., No. 13, 1912.

Kelman, S. R. Experimental Emphysema, Arch. Int. Med., xxiv, 333, 1919.

LeCount, E. R. Disseminated Necrosis of the Pulmonary Capillaries in Influenzal Pneumonia, Journ. Am. Med. Ass., lxxii, 1519, 1919.

Leriche, M. Emphysème traité par l'operation de Freund, Lyon méd., Vol. CXXII, pages 28, 604, 1090, 1284, 1914.

Lord, Frederick T. Diseases of the Bronchi, Lungs and Pleura, 9, 140, Lea & Febiger, Philadelphia, 1915.

Roubashow, S. La Théorie et l'opération de Freund dans l'emphysème pulmonaire, Revue de chir., xlviii, 417, 1913.

Staehelin, R. Pathologie, Pathogenese und Therapie des Lungenemphysems, Ergebn. d. inn. Med. u. Kinderh., xiv, 516, 1915.

Symmers, Douglas. The Pathological Similarity of the Pneumonia of Bubonic Plague and Pandemic Influenza, Journ. Am. Med. Ass., lxxi, 1482, 1918.

Torrey, Robert G., and Grosh, Lawrence C. Acute Pulmonary Emphysema Observed During the Epidemic of Influenzal Pneumonia at Camp Hancock, Am. Journ. Med. Sc., clvii, 170, 1919.

Tuffier. Bull. et mém. Soc. de chir. de Par., xxxvii, 138, 1911.

——— Emphysème pulmonaire opéré depuis quatre ans., Bull. et. mém. Soc. de chir. de Par., xl, 244, 1914.

Velden. Die Behandlung d. Lungenemphysems, Therap. Monatsh., 1912, June, s. 469.

LUNG ABSCESS AND GANGRENE

PHILIP H. PIERSON

Beck. Ueber Lungengangren, Deutsche med. Wchnschr., xxxiii, 409, 1907.

Bruns, O. Entstehung des Emphysems, Berl. klin. Wchnschr., 1910.

Bruns-Sauerbruch. Künstliche Erzeugung von Lungenschrumpfung durch Unterbindung von Asten der Pulmonalarteria, Mitt. a. d. Grenzgeb., d. Med. u. Chir., xxiii, 343, 1911.

Cumstom, C. G. The Treatment of Pulmonary Gangrene in Children, Therap. Gaz., Detroit, 3d Series, xxxvii, 542, 1921.

Cutler, E. C., and Hunt, Alice M. Postoperative Pulmonary Complications, Arch. Int. Med., xxix, 449, 1922.

Fraenkel, A., and Körte, W. Present Status of Lung Surgery, Berl. Klin. Wchnschr., xlix, 241, 289, 347, 1912.

Freund, W. A. Ueber primäre Thoraxanomalien, speziell über die starre Dilatation des Thorax als Ursache eines Lungenemphysems, Berlin, 1906.

Garré. Die chirurgische Behandlung der Lungenkrankheiten, Mitt. a. d. Grenzgeb. d. Med. u. Chir., ix, 322, 1902.

——— Die Operation des starr dilatierten Thorax, von C. Garre-Bonn, Ergebn. d. Chir. u. Orthop., Vierter Band, 265, 1912.

Hedblom, C. A. Pulmonary Suppuration. Med. Rec., xcvi, 441, 1919.

Jackson, C. Prognosis of Foreign Body in the Lung., Journ. Am. Med. Ass., lxxvii, 1178, 1921.

Kissling. Ueber Lungenbrand, Mitt. a. d. Hamb. Staats-Krankenanst., 1906.

Laignel-Lavastine and Couland. Abcès du poumon à pneumocoques, Bull. et mém. Soc. méd. d. hôp. de Par., 3d S., xlv, 1195, 1921.

Lenhartz. Handb. d. spec. Therap., Pentzoldt-Stinzung, 1910.

Lilienthal, H. Resection of the Lung for Suppurative Infections, Ann. Surg., lxxv, 257, 1922.

Norris, F. A. Postoperative Pulmonary Complications, Illinois Med. Journ., xl, 288, 1921.

Passler and Seidel. Beitrag zur Pathologie und Therapie des alveolaren Lungenemphysems, 1907.

Perthes. Zur operativen Behandlung des chronischen Lungenabszesses, Verhandl. d. deutsch. Gesellsch. f. Chir., xxxvii, 214, 1908.

Quincke. Zur operativen Behandlung des Lungenabszesses, Berl. klin. Wchnschr., No. 19, 1887, No. 18, 1888.

——— Ueber chirurgische Behandlung der Lungenkrankheiten, Mitt. a. d. Grenzegeb. d. Med. u. Chir., ix, 305, 1902.

Rathery, F., and Cambessédès. Traitement de la gangrène pulmonaire par la sérothérapie, Bull. méd. Par., xxxv, 795, 1921.

Sauerbruch, F. Die Chirurgie der Brustorgane, 521-536, 2d Ed., i, Berlin, Springer, 1920.

——— Die chirurgische Behandlung der Lungenkrankheiten, Spec. Path. und Therap. inn. Krankh., 343, 1921.

Tuffier. Bull. et. mém. Soc. chir. de Par., xxxvi, 529, 1910.

Von den Velden. Der starr Dilatierte Thorax, Stuttgart, 1910.

Wessler, H. Lung Abscess and Bronchiectasis, Am. Journ. Roent., vi, 161, 1919.

Whittemore, W. Lung Abscess, Surg. Clin., North America, Philadelphia, i, 765, 1921.

Tumors of the Lung[1]

George Blumer

Flint. Yale Med. Journ., Feb., 1910.

Meltzer. Med. Rec. N. Y., March 19, 1910.

Meyer. Ann. Surg., July, 1910.

——— Journ. Am. Med. Ass., Dec. 11, 1909.

——— N. Y. Med. Journ., lxxvii, 1910.

Robinson. Surg., Gynec. & Obst., March, 1910.

——— Boston Med. & Surg. Journ., Dec. 8, 1910.

Sauerbruch and Robinson. Ann. Surg., March, 1910.

Seydel. München. med. Wchnschr., lvii, 452, 1910.

---

[1] In this list the articles quoted are usually those containing references to other literature.

CHAPTER VI

DISEASES OF THE PLEURA

JOSEPH A. CAPPS

Historical.—The term "pleuritis" was used by the old Greek physicians to designate those inflammatory affections of the respiratory organs in which pain in the chest was present. Hippocrates distinguished pleuritis from peripneumonia and classified the varieties according to location into P. ascendens and descendens and P. anterior and posterior; according to cause into P. sanguinea, biliosa, pituitosa, and melancholia; according to the character of the exudate into P. sicca and humida. Herophilus and his followers believed the lung is always inflamed in pleuritis, but that the pain originated in the costal pleura and intercostal muscles.

The view that pleuritis is only a complication of lung inflammation was also held by writers of a more recent period (Sydenham, 1749). Pinel was one of the first (1818) to create for pleuritis the position of an independent disease of the serous membranes. It was reserved for Laënnec (1825) to build the foundations of the pathological anatomy of pleuritis, and by his discoveries and observations in auscultation and percussion to develop the science of diagnosis in diseases of the chest. As a result of his teachings many contributions to the subject have been made, chief of which in importance ranks the universal treatment of pleural effusions by thoracentesis.

Thoracentesis.—Paracentesis of the chest for the purpose of withdrawing fluid exudate has been practiced from the earliest periods. The history of this operation from its inception in ancient times, through centuries of neglect, to its revival in the nineteenth century is one of the most interesting chapters in medical therapy. What a remarkable tribute to the influence and dominance of the Greek school of medicine that for over two thousand years its teachings and tenets were accepted as authoritative and final!

Hippocrates advocated paracentesis for the identical conditions in which it is practiced to-day, namely, for effusions of every kind whether they be simple dropsy, purulent hemorrhagic exudates, or a combination of fluid and air. The last-named condition of hydropneumothorax he was especially successful in treating, because he had discovered a reliable sign for its recognition in the so-called succussion sounds. Doubtless better results in the treatment of the other forms of pleural exudates would have been obtained had he been able to make accurate diagnoses. As a consequence of this uncertainty in recognizing simple effusions, however, tapping was resorted to only when enormous distention of the chest had taken place, with a bulging of the interspaces from the pent-up pus, or when a traumatic pleurisy with pneumothorax had occurred. The opening was made by bistoury or actual cautery either through an intercostal space or a perforated rib. The fluid was allowed to flow until the cavity was empty, and, if necessary, the opening was cleared out from time to time with a metal sound.

Galen and Celsus accepted these Hippocratic teachings, but apparently had little experience with the procedure.

During the Dark Ages paracentesis received very little attention, except as to the relative merits of cautery and knife. In the seventeenth century Goulu (1624), Lusitanus (1644), and other surgeons who were performing abdominal paracentesis advocated tapping for hydrothorax as well as for empyema and claimed favorable results. The danger from air entering the chest during the evacuation of fluid was the subject of many polemics at this period. Bartholin observed that in penetrating wounds of the chest recovery was most rapid when the wound was closed, and from this he reasoned that the opening made in paracentesis should be closed as soon as possible. Bontius, on the other hand, looked upon air in the chest as harmless. This fear of pneumothorax, however, increased and led to the introduction by Scultetus (1669) of evacuation by aspiration and suction. Not long afterward Drouin (1675) and Lurde (1765) recommended for thoracentesis the use of the trocar, such as was employed in abdominal puncture, but the instrument was considered too crude by the leading physicians of that day. The chief objections to the trocar were the danger of penetrating the lung, of injuring the intercostal artery, and, finally, of withdrawing the fluid too rapidly. To obviate the last-mentioned difficulty only a small portion was drawn off every day. With the advent of Laënnec came a new order of things. Speculation gave way to careful observation; guessing to exact diagnosis. It became possible to detect the presence of pleural effusion and to locate it. Nor was Laënnec slow to follow up his pathological and clinical discoveries with logical suggestions for treatment. With rare discernment he stated that acute pleuritis with rapidly forming exudate offered a better prospect of cure by tapping than the chronic cases of empyema. His experiences in draining empyema by the trocar met with little success. Strange to say, Laënnec's own countrymen were slow to adopt the operation, since it was nearly twenty years later that Trousseau read his celebrated memoir before the academy, and by his influence and extensive experience established thoracentesis as a safe and valuable method of treatment. In England the innovation was brought about by Davis (1835), Roe (1844), and Hughes (1846). Likewise, in Germany, Becker (1834) and, in Austria, Schuh and Skôda (1841) were the champions of the procedure.

America is indebted to the writings and observations of Bowditch for the introduction of this treatment. He relates in 1848 that "the general voice of this country is against operation in any case, and that when advised at all the operation is incision"; Wyman improved the old trocar by devising a smaller instrument which was connected with an aspirating pump. It was this apparatus that Bowditch employed. The indications given by Bowditch are to tap whenever the chest is full, in either acute or chronic cases; in cases where there is alarming dyspnea even if the quantity of fluid is small; in all acute cases where absorption is slow.

The introduction of aseptic principles has had its effect in minimizing the dangers of infection and has thereby served to popularize the operation. Otherwise no important advance has been made in this form of treatment except in the improvement of the apparatus.

PLEURISY

Clinical Pathology.—In order intelligently to assist nature in the treatment of pleuritis it is necessary for the clinician to have a clear understanding of the nature of the inflammatory process, to discover, if possible, its origin, to comprehend as fully as may be the physical, chemical, and physiological steps by which spontaneous absorption and resolution takes place.

In pleuritis there is always an exudate which is composed of blood-cells and liquid. When the inflammation is very mild the pleural lining is preserved, but when it is severe a thick layer of fibrin forms, and the endothelial cells perish in the process of coagulation necrosis. There are many varieties of exudate, but the chief forms are the fibrinous, or dry, pleurisy; the serofibrinous, in which a considerable amount of clear fluid is present; the hemorrhagic; and the purulent, in which the fluid is cloudy and thick from the presence of pus cells.

Absorption of Exudates.—For the few days of the inflammation secretion is actively carried on by the endothelial cells, and under the influence of chemotaxis the leukocytes are poured out by the blood to participate in the fibrin formation. The outflow of serum from the blood is largely regulated by osmosis.

When the irritation ceases and the inflammation subsides absorption begins.

Absorption of Fibrin.—The absorption of fibrin is preceded by a gradual disintegration and solution of the solid material. Opie has shown that the polynuclear leukocytes produce an enzyme, called leukoprotease, which in an alkaline medium digests the fibrinous substance and thus prepares it for absorption. At the same time, new blood-vessels make their way from the serous layer into the coagulated mass and the *organization* of connective tissue is inaugurated. In the course of time the operation of these three agencies—digestion, absorption, and organization—brings about the disappearance of the thick false membrane and leaves a thinner layer of tough fibrous tissue. This scar tissue remains indefinitely as an adhesion.

Absorption of Fluid.—The absorption of fluid exudates is more simple and expeditious. Under favorable circumstances the liquid may disappear entirely in two or three weeks, but with surprising frequency the fluid may show little or no tendency to diminish for a long period of time. What is the reason for this failure on the part of the system to rid itself of a harmful secretion? As the exudate is carried away by the lymphatics and blood-vessels, it is apparent that any mechanical obstruction of these channels would seriously interfere with its removal.

The *lymphatics* principally concerned in the drainage of pleural exudates are those situated in the costal and mediastinal pleura. Fleiner injected India ink into the pleural cavity and found the pigment was taken up by the parietal pleura and deposited in the intercostal lymph glands; also by the mediastinal pleura and deposited in the tracheal lymph glands, but never in the bronchial glands. The diaphragmatic pleura did not absorb pigment. Interesting in this connection is the observation that pigmented fluid in the abdominal cavity is transported to the lymphatics of the diaphragm but *does not reach the pleural cavity.* The observations of Karsner and Swanbeck after injecting carmine and lamp black in the pleural cavity seem to show that the particles are taken up by the endothelial cells in situ rather than as free pigment. The endothelial cells lining the parietal pleura exhibited more phagocytic action than those of the visceral pleura.

The efficiency of lymphatic drainage depends not only on the patency of the ducts, but also on the respiratory movements of the thorax. Dybkowsky claims that the antagonistic action between lungs and intercostal muscles during inspiration is of great importance in absorption. In rarefied air inspirations are more forceful, the intercostal spaces are depressed, and absorption is rapid. In compressed air, on the other hand, the respiratory movement is limited and absorption is slow. For the same reason a large collection of fluid tends to immobilize the affected side of the chest and thereby interferes with the lymphatic circulation.

Absorption of pleural fluid by the *blood* depends to a large degree on osmosis. Starling and Tubby injected 50 c.c. of a 10 per cent solution of NaCl (hypertonic) into a healthy pleural cavity, and three hours and a half later found 170 c.c. of fluid in the chest, thus demonstrating the osmotic flow of liquid *into* the pleural cavity. When they injected a hypotonic solution (NaCl, .75 per cent, or distilled water) into the thorax absorption rapidly took place, thereby showing the osmotic flow *out* of the pleura. An isotonic solution (NaCl, .92 per cent), however, was absorbed with a rapidity equal to that of the hypotonic solution, a fact that cannot be explained by osmosis. It is necessary to assume, therefore, that the endothelial cells are directly active in absorption and that when they have reached their limit the passage of fluid is determined by differences in osmotic pressure.

Another factor influencing absorption is the *intrathoracic pressure.* In the light of the experimental work of Emerson it is probable that absorption is favored by moderate pressure of fluid within the thorax. Excessive pressure exerted by a large accumulation of fluid impedes the return of the venous blood to the heart through the inferior vena cava and thereby diminishes the systolic output of the heart and lowers the systemic blood-pressure.

Purulent exudates may undergo absorption, but unless evacuated by artificial means they often digest the surrounding tissues and rupture through the bronchial tubes or the chest wall. Encapsulation and caseation are occasional end stages of the spontaneous cure.

Absorption of Gases.—Rosenbach established the fact that O and CO_2 are more quickly absorbed by the pleura than Nitrogen. A Schmidt, after injecting 1,000 c.c. of O into a healthy pleural cavity, found that absorption was complete in three to six days. Air required six to twelve days. He repeated these experiments in cases of pleurisy, and noted that the absorption of all gases as well as fluids was much delayed. Evidence of the gas could be detected by percussion weeks after its introduction.

The absorption of gas is interfered with not only by inflammation of the pleura, but also by a high intrapleural tension which restricts the movements of the lung and the diaphragm. For this reason gas is more slowly absorbed when the amount is excessive or when a large amount of fluid is present.

Permeabilty of the Mediastinum.—In contrast to the diaphragm the tissues of the mediastinum are highly permeable. If normal salt solution is injected into the pleural cavity of a dog in liberal quantities a large proportion makes its way through the mediastinum to the opposite pleural cavity before absorption takes place. Cottonseed oil is absorbed very slowly by the pleural membrane, but it has no difficulty in penetrating the mediastinum.

Clinical experience likewise indicates that transudates of mechanical origin (for example, cardiac dropsy) shift readily from one side of the chest to the other. Pleuritic exudates of inflammatory origin, however, do not display any such tendency. The exudate remains localized on one side. We must, therefore, assume that the serous inflammation destroys the permeability of the mediastinal membrane.

Mechanical Effect of Exudate.—Garland was one of the first to demonstrate that pleural fluid first collects at the base of the chest between the under surface of the lower lobe and the diaphragm. A large quantity may collect in this region before dulness is apparent. The fluid compresses the lung in an upward direction, and by its weight tends to depress the diaphragm. The mere obliteration of lung space, however, is a less serious event than the impairment of respiratory action owing to the presence of a foreign body between the intimately opposed surfaces of lung and diaphragm. With further accumulation of fluid the lung retracts still more, until it usually lies close to the spinal column. By this time the diaphragm is flattened out and occupies a low position and ceases to move on the affected side with respiration. Similarly the intercostal muscles on the affected side are inactive. The heart is displaced toward the healthy side, and the mediastinum is pushed over, especially at the lower levels.

The retracted lung becomes atelectatic, and unless relieved of the pressure of fluid may be so bound by adhesions that it cannot reëxpand, or, at best, only partially.

The Intrathoracic Pressure of Exudates.—Quincke measured the tension of pleural effusions and found an absolute pressure varying from 0 to 26 mm. Hg, with an average of 10 mm. The pressure, as a general thing, is proportionate to the amount of fluid present within the thorax. The positive pressure tends to be neutralized, and may even become negative during the act of inspiration. The more mobile and expansile the chest wall, and the more active the opposite lung, the greater is the tendency to a low positive or to a negative pressure. For this reason the exudate in children is under a lower pressure than in old people. The presence of adhesions by immobilizing the ribs gives rise to a higher pressure of an effusion. Schreiber is of the opinion that the difference in pressure of the effusion between expiration and inspiration, or the *fall* in pressure during inspiration, is a reliable measure of the compensating activity of the healthy lung. Furthermore, he considers a strongly negative pressure after tapping an evidence of normal expansion of the well lung and, therefore, a favorable prognostic sign.

Influence of Exudates on Circulation.—If fluid is injected into the pleural cavity of an animal the general *arterial pressure* maintains a constant level until the space is almost filled, then quite suddenly the pressure falls to a low point and death is imminent. Lichtheim's experiments teach us that four-fifths of the lung vessels can be excluded from the circulation by ligatures without causing any marked change in the *systemic* arterial pressure. This is by no means a proof, however, that the various parts of the body are well supplied with blood. The pressure in the *pulmonary* artery is increased even by a moderate effusion, and the preservation of a normal tension in the systemic arteries is merely an evidence that the right heart is capable of performing its additional work.

Accumulation of fluid in the chest not only impedes the flow of blood through the lungs, but by lowering or destroying the normal negative pressure within the thorax obstructs the flow of blood from the body veins. Under normal conditions each inspiration creates such a strong negative pressure that the blood is sucked into the thoracic veins from the peripheral veins. The presence of fluid impairs this inspiratory flow, and the pressure in the *body veins* steadily rises. Usually the venous stasis is partly overcome by compensating inspiratory movements, that is, by deeper inspiration.

It should be emphasized that the mere obliteration of lung space is of little consequence. More serious in their effects are the obstruction of the pulmonary circulation, which is compensated for by the right heart, and the impairment of the negative intrathoracic pressure, and resulting stasis in the body veins, which is compensated for by deepened respiration.

Cause of Death from Pleural Exudate.—Many explanations have been advanced for the occurrence of sudden death in cases with a large pleural effusion. Trousseau and Leichtenstern supported the view that *heart failure,* induced partly by the high pulmonary tension and partly by displacement and tension of the organ, was the cause. In order to accept this view we must suppose that the heart muscle has been greatly weakened by the toxins of disease. Yet experience teaches that sudden death often takes place from a rapid collection of fluid long before the infection could badly cripple the heart. Moreover, the researches of Romberg and Pässler have given ample proof that the heart can successfully resist infectious toxins for long periods, and that terminal collapse marks a breakdown on the part of the vasomotor system rather than of the heart. Rosenbach attributes the fatal collapse in pleural effusions to an obstruction of the venous flow from the body into the thorax. A contributing factor is produced by the violent action of the diaphragm on the healthy side, which twists and perhaps kinks the inferior vena cava. Quite apart from any kinking of the vein, there seems to be abundant reason for accepting this view. The progressive obstruction to the venous flow finally brings such a small volume of blood to the heart that the systolic output is inadequate to fill the arteries, and the general pressure rapidly falls.

It is nearer the truth to say that the *venous circulation fails* to supply the heart with blood than to say the heart fails.

Pain Sense in the Pleura.—Pain is perhaps the most constant and important symptom of pleuritis. A careful observation of the distribution of pain is essential to a correct diagnosis of the seat of inflammation.

In an experimental and clinical study of pleural pain, we have reached the following conclusions:

1. The *visceral* pleura is not endowed with pain sense.

2. The *parietal* pleura is richly supplied with sensory fibers from the intercostal nerves. Irritation of the pleura induces sharp pain that is *accurately* located by the individual over the spot that is touched. Such irritation in our experience never gives rise to "referred pain" in the neck or in the abdomen. The power to locate sensory impressions seems more highly developed in the anterior and lateral portions of the parietal pleura than in the posterior.

3. The *diaphragmatic* pleura derives its sensory supply from two sources, the phrenic nerve and the last six intercostal nerves. The *central* portion of the diaphragmatic pleura is innervated by the phrenic nerve. Irritation of this portion sets up pain in the neck. A peripheral rim of the diaphragmatic pleura, which is 2 or 3 inches wide anteriorly and laterally, and a segment corresponding to the posterior third of the membrane, are innervated by the sensory fibers of the *last six* intercostal nerves. Irritation of these areas gives rise to pain in the lower thorax, in the lumbar region, or in the abdomen.

The *neck pain* arising from irritation of the central portion of the diaphragmatic pleura is a true referred pain, the afferent impulses reaching the cervical cord through the phrenic nerve trunk and exciting efferent impulses in the skin and superficial tissues supplied by the third and fourth spinal segments. This pain is characterized by a point of maximum pain and tenderness and by a surrounding zone of cutaneous hyperesthesia and hyperalgesia. The maximum point of pain has a remarkable tendency to appear along the ridge of the trapezius muscle (fourth spinal segment).

The pain elicited by irritation of the peripheral or posterior portion of the diaphragmatic pleura is also a true "referred pain." The pain is usually distributed in segmental areas over the lower thorax and epigastrium, sometimes extending downward over the whole abdomen on the same side (seventh to twelfth dorsal segments). The pain is associated with hyperesthesia and hyperalgesia of the skin and superficial tissues on pressure.

The *distribution* of the pain is determined by the degree of irritation and by the part that is touched. The more intense the irritation of the pleura, the greater the tendency of the pain to spread down over the lower abdomen. Rarely from strong pressure the pain is *bilateral,* extending over both sides of the abdomen. When the posterior portion of the diaphragmatic pleura is irritated the pain is most marked in the lumbar region.

4. The *pericardial pleura* receives its sensory innervation chiefly if

not exclusively from the phrenic nerve. Irritation of this part of the pleura is followed by pain in the neck of the same character and in the same locations as that induced by irritation of the central portion of the diaphragmatic pleura. This pain is a "referred pain," characterized by a maximum point of tenderness and pain, by hyperesthesia and hyperalgesia to pressure of the surrounding skin, and by muscular rigidity.

5. In an extensive series of cases of diaphragmatic pleurisy in some of which the diagnosis was confirmed by autopsy, pain of the segmental type over the lower thorax, abdomen, or lumbar region was present in the great majority. In about one-half the cases pain in the neck was complained of. This cervical pain was always in the region supplied by the third and fourth spinal segments, showing a preference for the trapezius ridge. There was always a point of maximum pain and a surrounding area of hyperalgesia and hyperesthesia of the skin.

Onset of Pleurisy.—The onset of pleurisy is so protean in its manifestations that only a rough picture can be drawn. Two types may be described:

1. The attack begins abruptly with chilly sensations or an outspoken chill followed by fever, pain, sweating, and cough. Such is the origin of pleurisy accompanying pneumonia or of a primary pneumococcus infection. Many tuberculous pleurisies also have a stormy onset.

2. The initial stage is insidious, often without any symptoms pointing to the chest. A rapidly developing anemia, the appearance of a digestive disturbance with loss of appetite and weakness, or of shortness of breath on exertion, lead to a physical examination and the discovery of fluid in the pleural cavity. The pleurisy can develop de novo or come on as a complication of a chronic disease, such as cancer or Bright's disease.

Clinical Course.—The further course and prognosis cannot be predicted from the nature of the onset, for an acute stormy attack may progress favorably and end in complete spontaneous absorption and recovery, whereas the slowly forming exudate with little febrile disturbance sometimes displays a remarkable persistence and a tendency to reaccumulate even after evacuation. The helpful factors in determining the course of the affection are to be found in the underlying cause and in complications. A pneumococcus or tuberculous exudate is likely to absorb unless it becomes purulent, whereupon artificial drainage is imperative. Occasionally from a pneumococcus pleurisy a pneumonia starts up and places the subject in jeopardy. In tuberculous pleurisy a secondary involvement of the lung or of the bronchial glands is not uncommon and the outlook is rendered more grave.

A pleurisy complicating carcinoma usually results fatally, because it tends to reappear after repeated tappings. On the other hand, the pleural effusion complicating rheumatic arthritis nearly always ends in recovery.

Empyema, whether primary or a sequel to a serous exudate, is a dangerous condition which, as a rule, ends in fatal sepsis when left to nature. In rare instances, however, absorption takes place or spontaneous rupture occurs into a bronchus or externally through an intercostal space.

End Result after Absorption.—The end result in cases which recover under natural means is often a matter of surprise. A year after the attack an examination will reveal, perhaps, no evidence of thickening and a complete return of function on the part of the lung. In a less favorable case some retraction of the chest on the diseased side is found, and a moderate restriction of the respiratory excursion. In more chronic and obstinate cases, particularly those with purulent exudate, the side of the thorax is flattened, the ribs and diaphragm move very little with respiration, the percussion note is dull from the thickened pleura, the lung is functionless, and the heart displaced. Needless to say the last-described termination can generally be obviated by modern methods of drainage.

Treatment.—Prophylaxis is a term that can be used only in a relative sense in connection with pleurisy. In an infectious disease like typhoid fever definite preventive measures to safeguard water and food are truly prophylactic. The possibility of carrying out prophylactic precautions in pleurisy resolves itself into an effort to prevent the primary disease to which the pleural inflammation is secondary, or to avoid those exciting influences which induce the complication of pleurisy. Thus a phthisical individual should be more careful than a healthy one in avoiding exposure to cold, overexertion, or exercises that subject him to trauma. The same warning is necessary for the subject of chronic heart or kidney disease.

A causal therapy also has its sharp limitations, since for most forms of pleuritic infection no specific treatment has been perfected. It is true that some authors speak of the salicylates as specific for the rheumatic forms of pleurisy, but it cannot be fairly claimed that salicylates are a cure for the ordinary rheumatic arthritis. More reasonable is the belief in the specific action of the iodids in syphilitic pleurisy. Unfortunately these cases are not numerous and always are difficult to recognize. The last few years have seen a growing tendency to search the body over for a primary focus of infectious material, and to eradicate this source of blood contamination, whether it be a middle-ear abscess, a carious tooth, or a pus pocket in the tonsil. Feidler in 1895 called attention to the frequency of pleurisy following tonsillar infection, either alone or associated with arthritis. The removal of such a focus by tonsillectomy has given many brilliant results and is deserving of more frequent trial in the future.

General Indications—*Rest.*—Regardless of the special origin of the pleurisy, rest is the first essential of treatment. There is no doubt that rest conserves the forces of the body organism and thereby increases its resistance against the invading poison. When the onset is acute, with fever, the patient should be confined to bed and remain there until the temperature has returned to normal. The patient often fails to realize the necessity of complete rest. Hence it is incumbent upon the physician to explain the danger of extension of the process from pleura to lung, and especially of the development of a chronic tuberculosis. Physical exercise does not simply increase the work of the heart, it also heightens the respiratory movements and directly irritates the inflamed tissues.

Fresh Air.—Fresh air is of great benefit in pleurisy, as it is in all other affections of the respiratory apparatus. Whenever possible, a sleeping porch or tent should be utilized. The patient out-of-doors breathes more slowly and with greater ease and secures sounder sleep. Why a person is more comfortable out-of-doors than in a room with open windows is not generally understood, although the testimony of tuberculous patients to the fact is overwhelming. Hill and Pflüge have shown by experiments that indoors the expired air, as a rule, remains for some time in the vicinity of a person's face, so that he inhales air containing a considerable proportion of his own breath. They find that the chemical changes in the character of this reinspired air, such as an excess of carbon dioxid, or deficiency of oxygen, exercise no deleterious effect on the individual. The sensations of discomfort are due to the heat and moisture present. Out-of-doors, on the other hand, there is almost always sufficient shifting of air currents to quickly carry the expired breath away. In other words, motion of the air combats the harmful effects of heat and moisture thrown off by living bodies, and is therefore necessary for perfect ventilation. The use of the electric fan in crowded wards or in the sick room should be more generally adopted.

FIBRINOUS PLEURISY

The fibrinous, or "dry," pleurisy is the most common variety. Horder says:

> "There are probably few diseases more often diagnosed without adequate reason than pleurisy; there are few diseases that exist more often unsuspected."

It is probable that many instances of so-called intercostal neuralgia or rheumatism are associated with a limited fibrinous exudate which may entirely escape the attentive ear of the physician. To designate these casual adhesions as invariably tuberculous is beyond the warrant of our knowledge. E. Grawitz believes that many of these adhesions are due to the inhalation of particles of dust, which find their way through the lymphatics to the pleura. Apical pleurisy is often the first manifestation of pulmonary tuberculosis and hence important to recognize as early as possible. Sergent has called attention to the syndrome of pupillary irregularity and supraclavicular adenitis as a help in the diagnosis. The adenitis is found in the angle formed by the internal extremity of the clavicle and the sternal insertion of the sternomastoid muscle. The glandular swelling seems to be associated with inflammation of the apical pleura rather than with the lung itself.

The treatment of acute fibrinous pleurisy of an outspoken type is largely symptomatic, the etiological factors having much to do with the later management of the case.

Acute Fibrinous Pleurisy.—The symptoms of acute dry pleurisy are *pain,* often described as a "stitch in the side," cough that is sharp and unproductive, rapid and *shallow respiration,* and fever. The pain is aggravated by cough or deep breathing, and is most intense in the region where the friction rub is heard with the stethoscope.

Pain.—Strapping the affected side with a broad strip of adhesive plaster sometimes brings relief by lessening the excursion of the ribs. In our experience, however, the procedure is not often successful and has the disadvantage of being a hindrance to auscultation.

Hot and cold applications to the seat of the pain are both employed. A hot compress or hot-water bag is usually grateful to the patient. Cold, in the form of an ice-bag or a compress, is preferable, since it acts favorably on the fever. A remarkably efficacious way of applying cold for the relief of pain is by the use of strips of linen, cut 12 inches long and 2 inches broad, first laid on a cake of ice and then applied parallel to the ribs. The strips should be frequently removed and replaced with others freshly chilled by ice. By this method the author has seen the severest pain gradually subside in the course of a few hours.

Opiates are valuable and at times indispensable. At the onset, with febrile symptoms, 10 gr. (0.6 gm.) of Dover's powder, or 1/6 gr. (0.01 gm.) of morphia, is helpful. The morphin may be repeated the following night if the pain persists. At the same time, the cough is benefited by this narcotic.

The bowels should be cleared out at the onset with calomel, followed by a saline. It is desirable to continue the use of Epsom salts or Hunyadi water every morning until the fever subsides. Under these measures the ordinary case of pleurisy makes a speedy recovery. For several weeks after the disappearance of all symptoms especial care should be taken to avoid chilling, violent exercise, or great fatigue.

Serofibrinous Pleurisy.—The treatment of pleurisy with effusion resolves itself into four lines of effort: (1) the treatment of the infection itself; (2) the relief of distressing symptoms; (3) the attempt to limit the secretion and encourage its absorption; (4) the evacuation of fluid by operative means.

Treatment of the Infection.—At the onset of an acute attack much may be done to modify its severity by the prompt administration of calomel, followed in the course of eight hours by a full glass of Hunyadi water or an ounce of Epsom salts. If fever sets in abruptly a hot bath is advisable to promote diaphoresis. Medicinal remedies with a specific germicidal action on the infectious agents are wanting. Since Feidler showed that pleurisy often has the same infectious origin as rheumatic arthritis, many writers have advocated large doses of the salicylates as an efficient antiseptic. Rosenbach advocates this remedy because the derivatives of the salicylates are found after internal administration in pleural exudates and transudates.

That salicylic acid reduces the temperature and notably alleviates pain is not open to question, but we believe with Unverricht that the clinical data at hand do not justify the statement that it materially changes the course or duration of the disease. It is best given in the early stage and in large doses (40 to 100 gr. a day—3.0 to 7.0 gm.).

Quinin is considered by many to influence the disease favorably. Carrière recommends giving 5 to 10 gr. (0.3 to 0.6 gm.) per diem throughout the febrile period.

Symptomatic Treatment.—The relief of *pain* is obtained by the measures described for dry pleurisy. Since the pain is usually severe and lasts but a few days the injection of *morphin* is altogether the most effective remedy. Next in value is the use of *cold applications,* or, in weak individuals with poor circulation, of hot compresses.

Dry Cupping.—Dry cupping over the painful area acts as a derivative, withdrawing blood from the congested pleura. It is a simple procedure and often yields excellent results.

Painting.—Painting the skin with tincture of iodin has less to recommend it. Inunction of the skin with oils containing salicylic acid has been defended, because absorption undoubtedly takes place. In order to bring about a considerable absorption, however, the rubbing must be continued for twenty minutes or more, and this is a trying experience for the patient.

Cough.—The cough is at times a troublesome symptom, as it increases the pain. As before stated, small doses of morphin or codein, gr. 1/4 (0.02 gm.), can be given often enough to afford relief.

Fever.—Fever is seldom so high as to demand vigorous means for its reduction. Salicylate of soda or aspirin is preferable to antipyrin, phenacetin, and other coal-tar derivatives, as the latter exert a depressing effect upon the heart and circulation. Sponging the body with cool water or with alcohol and water is perhaps the best method of lowering the body temperature. Some prejudice exists against cool baths in pleurisy, but the success of this form of hydrotherapy in pneumonia is the most convincing answer to criticism.

To Check Secretion and Aid Absorption of Effusion.—The *dry diet* of Schrott is a popular therapeutic practice in Germany. This consists of lean roast veal, stale rolls, and only sufficient water for deglutition for the first three days; then a half pint of red wine is permitted, and by the end of the week is increased to a pint. The custom of *limiting the liquids* ingested has many adherents, but is not altogether supported by physiological reasons.

Transudates and Exudates.—Transudates are of non-inflammatory origin and represent the outpouring of serum into the body cavities. They are caused by mechanical obstruction of the veins—venous stasis, whereby the serum escapes into the tissues (diapedesis)—or by chemical processes effecting an outflow of serum (osmosis). In transudates the blood-vessels and lymphatics remain patent. Hence it is easy to understand that all measures tending to reduce the body fluids (catharsis, diuresis, bleeding, etc.) have a direct influence on the transudate and promote its absorption by lymphatics and blood-vessels. The evacuation of a large quantity of ascitic fluid in cardiac dropsy leads to a rapid absorption of pleural transudate. The complete disappearance of pleural transudate in two or three days is often seen, also, after a great outflow of urine or a watery diarrhea.

Exudates, on the other hand, are for the most part a secretion of the endothelial cells, and the quantity of fluid thrown out depends not primarily on stasis or on osmosis, but on the degree of irritation of the cells and their inflammatory reaction. An exudate can form even when the body is exsiccated by loss of fluids, as, for example, in dysentery. The small blood-vessels and lymphatics are usually obstructed by the inflammation.

Thus it happens that the removal of body fluids by any of the abovementioned methods does not necessarily bring about rapid absorption. Absorption of an exudate seems to depend rather on some reaction in which the serous cells begin to absorb instead of secrete, and perhaps, too, on the activity of proteolytic enzymes by which the lymphatic ducts and capillaries are cleared of their coagulated contents.

With a full knowledge of these facts it must be recognized, however, that an effusion may be a combination of exudate and transudate. Furthermore, it is possible that even in a pure exudate some of the lymphatics may remain permeable and respond in some degree to depleting measures.

Cathartics.—Hydrogogue cathartics, such as the compound jalap powder, 2.0 to 4.0 gm. (ʒss to ʒi), taken at bedtime, or a saline quaffed on rising, are more useful in relieving abdominal distention and, indirectly, the dyspnea than in promoting absorption of the fluid. One should be careful to avoid the exhausting effect of prolonged and excessive purging. A daily morning dose of sodium phosphate (ʒii—8.0 gm.) in a cup of hot water throughout the illness admirably serves the purpose.

Diuretics.—Diuretics, so efficient in transudates of cardiac and renal origin, are of less value in pleural exudates. Not infrequently it happens that, while diuretics are being given, a copious flow of urine starts up and simultaneously the pleural fluid diminishes. Rosenbach considers the diuresis a result of increased absorption from the pleural cavity, a sequence that is observed when no medication is being carried out. Of all diuretics, caffein and diuretin [gr. xv (1.0 gm.) three times per diem] are the most reliable in their stimulation of renal cells.

Cardiovascular Stimulants.—These are indicated if the blood-pressure is low or if the heart shows evidence of weakness. *Digitalis* is most successful in those cases where, in the wake of an exudate, an additional transudate is created by a failing heart muscle. The improved heart action reduces the amount of fluid, and, at the same time, causes copious diuresis. Digitalis is best prescribed in the form of the tincture, 10 minims (0.6 c.c.) four times a day, or of the powdered digipuratum, gr. ¾ to 1½ (0.05 to 0.10 gm.) three times a day.

Caffein is valuable for a small weak pulse, as it raises blood-pressure. A convenient method of administration is the subcutaneous injection of the caffein and sodium benzoate, 1 gr. (0.065) every four to six hours.

Diaphoretics.—Diaphoretics have little to recommend them. Pilocarpin is especially objectionable because it stimulates bronchial secretion and thereby adds to the embarrassment of breathing. Drugs having some chemical property that promotes absorption of fluid have been extensively employed. Inunctions of mercurial ointment, as advocated by Meininger, are not recommended.

Iodids.—The iodids have long enjoyed the reputation of aiding absorption of exudates. Rosenbach states that iodids find their way into transudates but not into exudates. It has been claimed that iodids render the vessel walls more permeable and increase the activity of the leukocytes, but there is little evidence to back up this hypothesis. Holding to the belief that some pleurisies are of syphilitic origin, Steintzig looks upon iodid as a specific in these cases. Cushny contends that the undoubted efficiency of iodids in removing syphilitic gummata has led to the unwarranted belief that other forms of exudate are similarly influenced. How much of the prevalent faith in the absorptive power of iodids in serous exudates is based on mere tradition and how much on sound empiricism must be determined by future investigations.

When the exudate is absorbed and the fever has subsided the after treatment requires the greatest skill and judgment. The patient may be allowed to walk and indulge in other mild exercise. He should practice deep breathing, and spend a few minutes each day swinging light Indian clubs or dumb-bells in order to encourage deep respirations and the filling out of the retracted lung. Later on, rowing and swimming are ideal forms of recreation.

For at least one year following a pleurisy with effusion every effort should be made to live in the open air and sleep out of doors. Lord does not overstate the gravity of the condition when he says that "treatment should be based on the ascertained fact that three-fourths of all primary cases with effusions are due to tuberculosis and that two-fifths of primary cases develop some form of tuberculosis within four to six years." If the subject is a young person in school, he had best give up the confinement of study for one year and live in the country or on a ranch; if a clerk or other city worker with long hours at a desk, he had best change his employment to some task that keeps him in the open air. Of course among the poor it is not often possible to change employment or residence, but with little expense a sleeping porch can be added to the house.

Operative Measures.—Since the time of Trousseau, when thoracentesis had more enemies than friends, the operation has steadily grown in favor. To-day one seldom hears the question discussed as to whether tapping is justifiable. The interest in the subject is directed to the consideration of the most favorable time to operate and the best methods for its accomplishment.

Indications —*Indicatio Vitalis.*—The *indicatio vitalis* is the most firmly established of all. Trousseau was the first to realize that fluid may collect so rapidly that by its mere mechanical pressure it can cause death. He graphically relates several instances where in his earlier experience, with a large effusion accompanied by dyspnea, he timidly delayed tapping and needlessly sacrificed a life. In some acute cases the fluid accumulates with remarkable rapidity. Within two or three days the pleural cavity may be entirely filled. Rapid and labored breathing, orthopnea, and cyanosis are the symptoms which should warn the physician of danger. When physical examination reveals the upper border of fluid as high as the third rib in front, or the spine of the scapula behind, and rapidly rising, it is not safe to postpone paracentesis longer. Under these circumstances the pulse rate and pressure can be ignored, as they cannot be depended upon to forecast a dangerous or fatal collapse. The pulse is capable of maintaining a good pressure, though the volume usually diminishes, up to the breaking point. Then its tension quickly falls and death is imminent. The respirations, on the contrary, show signs of embarrassment at a much earlier stage, becoming rapid, labored, and at times irregular. If the fluid reaccumulates after tapping, evacuation may be repeated without detriment.

Large Effusions.—Large effusions of slower development must be withdrawn for the same reasons. Nearly every clinician with hospital experience can recall instances of large pleural exudate where thoracentesis was delayed on account of the absence of alarming symptoms and the patient suddenly died. It has been already mentioned that in *pleuritis acutissima* the pulse is of little value as a warning signal of danger. This is also true in the large effusions of slower onset. Furthermore, in these latter effusions even the respirations may be quiet and cyanosis be slight or absent, and yet danger lurks near by. The rapid development of alarming symptoms in these cases depends not on an exacerbation of the pleurisy with free exudate, but on a failure of absorption. Fluid may remain in the chest for weeks and months, and it is constantly working injury to various organs. The lung is impaired by pressure; the heart is at the same time hampered in its action and poisoned by the infection; the diaphragm is forced down by the weight of fluid and its tonus seriously impaired. The weakening of the diaphragm and intercostal muscles interferes with the mechanism by which, during inspiration, the venous blood is sucked from the body to the thoracic cavity, and this flow is further impeded by the presence of fluid in the chest, with its resulting positive pressure.

After struggling under this tremendous load for a long period, a trivial tax on the patient's strength, such as excitement, coughing, defecation, or slight muscular effort, may bring on circulatory failure. A safe rule is to remove the fluid if it has reached the level of the third rib anteriorly.

Moderate Effusion with Slow Absorption.—At the present day no one questions the propriety of operative procedure for the removal of a large collection of fluid or of fluid increasing at a rapid rate. Any disastrous result of expectant treatment in such cases can be attributed to either gross carelessness or procrastination. The patient with a moderate effusion that is absorbing very slowly runs less risk of sudden death but more risk of permanent injury. An effusion of moderate size, not requiring immediate evacuation, is often left to the care of nature. Weeks and months may elapse with no appreciable shrinkage in the volume of fluid. The patient may be walking about, short of breath and easily tired by exertion. Upon physical examination the presence of fluid can often be detected only by the most careful percussion, since the dulness may be attributed to the thickened pleura. Tapping and evacuation of the exudate in these cases are followed ordinarily by a rapid improvement in breathing and in general strength.

Other Considerations.—Other considerations that demand relief by tapping, quite apart from the amount of fluid present, are a marked displacement of the heart and other neighboring organs, and the development of dyspnea or cyanosis. A relatively small collection of exudate may be circumscribed and so situated as to cause a serious disturbance of the pulmonary circulation. The presence of myocarditis with dilatation of the heart, or an impairment of the respiratory function from an associated disease of the lungs, such as pneumonia, emphysema, etc., may necessitate the removal of comparatively small effusions.

Choice of Time for Operations.—For several days after the onset of an acute attack of pleurisy the fluid continues to accumulate. A time is reached when the inflammation subsides and the secretory activity of the pleural membrane becomes quiescent. This is the ideal time to perform thoracentesis unless immediate intervention is demanded by rapid or excessive accumulation. Generally a week or more elapses before the fever and secretory reaction decline. Since the time of Traube fever has been looked upon as a sign of inflammatory activity and a contra-indication to tapping. It is desirable, whenever possible, to wait for the temperature to fall, but Unverricht has challenged the attitude of indefinite waiting because of its presence. The existence of the exudate is perhaps a contributing cause to the fever; at any rate, evacuation frequently is followed by a fall in temperature, just as is commonly witnessed in emptying a pus cavity.

Contra-indications.—In the opinion of many authorities there are no contra-indications to thoracentesis in an outspoken pleural effusion. Rather would we speak of the desirability, in certain cases, to delay puncture and to withdraw a comparatively small quantity of fluid.

Königer has made a careful study of the influence of pleuritic effusion upon an underlying tuberculous infection. He finds the inflammatory reaction has a decidedly favorable effect on the course of the disease and often leads to spontaneous cure. The virtue of the pleurisy lies both in the compression and enforced rest of the lung and in the production of chemical antibodies, which tend to bring about immunity. Upon these grounds he advocates late tapping, preferably two weeks after the onset of a primary pleurisy. Moderate effusion is a secondary tuberculous pleurisy will often do more good than harm, and does not always need drainage. In an outspoken pulmonary tuberculosis with cavity formation the effusion may be allowed to remain indefinitely or, at least, only a small portion need be removed.

Location of Puncture.—The axillary space offers an excellent place for penetrating the chest wall, for in this region the muscular covering is thinnest and there is least injury inflicted upon soft parts. The intercostal spaces are wider here than posteriorly. The fifth and sixth intercostal spaces at the anterior or midaxillary line are usually chosen if the fluid is present in large amount. With a smaller exudate the posterior route is preferable, entering the sixth to the eighth interspace near the angle of the scapula or in the scapular line. One should bear in mind that a small effusion can frequently be drained posteriorly when a dry tapping is encountered laterally or anteriorly. As the fluid recedes the anterior portion of the chest becomes resonant, while posteriorly an area of dulness remains. For this reason a puncture in the back is more likely to yield a flow than one in front or in the side. Still another advantage of the posterior site is that the patient is not able to observe the operation and is less frightened. Unverricht considers the danger of perforating the diaphragm much less when the trocar enters the back than when it pierces the axillary space. For the inexperienced operator the posterior site is to be recommended on the ground of safety.

No rule can be laid down for the puncture of encapsulated exudates and for interlobar effusions. The location of the most outspoken physical signs of fluid, as determined by auscultation and percussion, must be the guide for the trocar. In obscure cases several places may be tried before the result is successful. Musser states that a circumscribed pleurisy is sometimes located by the presence of an overlying area of tenderness to pressure.

Position of Patient.—With many practioners it is still customary to have the patient assume a sitting posture either on the edge of the bed or astride a chair, with the head and arms resting on the back for support. In this way the back and axilla are well exposed, and by elevating the shoulders the interspaces are widened. The chief advantage of this position is based on the idea that good drainage can be secured only from the lowest point of the chest, where the fluid tends to gravitate. This reasoning is quite erroneous, for the rate of flow and the amount of fluid that escapes depend less on the hydrostatic pressure of the exudate than on the tension of the lungs and the activity of the intercostal muscles and diaphragm. A simple illustration of this fact is seen by the familiar observation that a large effusion often refuses to flow spontaneously, while a smaller effusion may gush out under high pressure.

Moreover, there are several objections to the sitting position, namely, muscular exertion is increased, especially on the part of the diaphragm; the tendency to cough is enhanced; the blood supply to the brain is diminished, thereby favoring faintness and vasomotor weakness. Indeed, syncope may occur.

The *recumbent position* with the shoulders raised by pillows or a lateral position with the affected side uppermost is recommended as a routine practice. The exceptions to this rule are cases of uncompensated heart disease, asthma or marked dyspnea, in which the upright posture is necessary for comfortable breathing.

Preparation.—Absolute cleanliness and attention to *aseptic* details should be rigidly enforced as in any other surgical operation. The instruments should be sterilized in boiling water, and care should be taken to see that the lumen of the trocar is patent. The skin is cleansed with soap and water followed by boiled water and alcohol. Over the immediate vicinity of the point selected for puncture the skin is then painted with tincture of iodin. We have found a very satisfactory disinfectant in the place of iodin to be a saturated solution of phenol, which is applied over a spot the size of a silver dollar and washed off in twenty seconds with alcohol. The phenol not only cleanses the skin but exerts an anodyne effect on the superficial nerves. Ethyl chlorid sprayed over the skin until freezing takes place also deadens the pain to some extent. The sharpest pain occurs not with piercing the cutaneous structures, but with that of the parietal pleura. Hence a deep injection of cocain is necessary to anesthetize all the tissues. Most clinicians find, however, that the preliminary injection of cocain is quite as distressing as the direct trocar thrust. General anesthesia should never be employed in tapping serous effusions, even in weak or hysterical individuals, on account of the danger of pulmonary congestion or respiratory failure.

Choice of Instrument.—Shall the trocar or hollow needle be used? This is a question that has elicited much discussion and that must be decided partly on the ground of personal preference. A simple hollow needle with a lateral foramen near the point can be introduced with less force and with less pain to the patient than a trocar. The lumen is easily cleared out by a stylet should any obstruction to the flow occur. The risk of tearing the lung with the sharp point, and of consequent production of pneumothorax or of bleeding into the pleural cavity, is more theoretical than actual, for such accidents are rare when one considers the innumerable times this operation is successfully performed. A more serious danger is encountered in the mere scratching of the visceral pleura toward the end of evacuation when the expanding lung comes in contact with the point with each respiratory movement. This irritation ordinarily gives rise only to cough, but exceptionally it may set up reflex disturbances of the heart and vasomotor system.

The *trocar* cannula, after the withdrawal of the cutting point, is less likely to irritate the visceral pleura and is, therefore, the safer instrument. In choosing a trocar it is well to select one of the largest size which can be readily inserted between the ribs, in order to provide for the free flow of a thick or stringy exudate.

Method of Evacuation.—The trocar inserted into the pleural cavity and the stilet removed, the fluid flows out spontaneously. The flow is intermittent, increasing with expiration and diminishing or even ceasing altogether with inspiration. A similar ebb and flow takes place in draining a small collection of ascitic fluid, but the variation is far less marked. There is always sufficient positive pressure in ascites to drain off most of the fluid. In a pleural effusion similar conditions do not always exist. If the effusion is very large the fluid is under sufficient positive pressure to expel the first portion in a continuous stream, then another portion during expiration only, then no more escapes, although the cavity may still contain two pints or more. If the effusion is of moderate or small proportions none may flow spontaneously because of the negative intrathoracic pressure. Under such circumstances aspiration is, of course, necessary.

Another phenomenon seen in draining the thorax, not observed in tapping the abdomen, is the tendency to suck in air during the act of inspiration and thereby to create a pneumothorax. This difficulty is overcome by attaching to the trocar a rubber tube about three feet long, which is filled with sterile water and allowed to carry off the pleural effusion by siphonage.

Aspiration.—Since the introduction of modern apparatus the aspiration method for evacuation of fluid exudate has become quite general. In skilled hands there is very little risk, and the operation can be completed expeditiously. For the inexperienced, on the other hand, the perfect control of the flow is a temptation to withdraw the flow too rapidly and to get "the last drop." The method of siphonage avoids these pitfalls, as the flow is necessarily slow and the cavity is never completely emptied. In the great majority of cases siphonage removes as much fluid as is desirable. If the spontaneous flow is too small, then a slow current can be established by means of gentle suction.

Exploration.—For the purpose of exploration it is common practice to employ a Luer syringe of 5 or 10 c.c. capacity, fitted with a long needle of large caliber. Sewall has devised a simple apparatus consisting of a needle connected by a line of rubber and glass tubing with the bottom of a bottle half-filled with water. From the upper portion of the bottle another tube passes through the rubber cork, and to this is attached a rubber tubing and mouthpiece. The muscles of the mouth serve as the piston, and the tip of the tongue as the valve of the syringe. It is simple, easily constructed, and can be used with a needle or trocar of any size.

Exploratory puncture to determine the presence of fluid and its location is often used in the office of the physician or at the bedside, with the deliberate intention of draining the cavity at a later time. When the diagnosis is reasonably certain the preliminary puncture should be dispensed with and the operator be prepared to withdraw the fluid at the first tapping.

The trocar should have a sharp stilet which fits tightly in the cannula. The cannula at its junction with the sharp point should be ground down to smoothness. The outer end of the cannula has two outlets, one with a stopcock for the admission of the stilet, the other placed laterally for the rubber connection, which need not be disturbed when the cannula is cleared out. By the siphonage method about three feet of rubber tubing leads to a water-containing receptacle. By elevating or lowering the free end of the tube a varying degree of suction is obtained. All connections should be air-tight, although the danger from the entrance of a moderate quantity of air has been exaggerated.

A good way to obtain a dependable aspirating force is partially to exhaust the air in a large bottle of a capacity of at least 2,000 c.c. One method of accomplishing this result is by setting fire to a small charge of alcohol in the bottle and quickly inserting the stopper before the flame is out. The rubber tubing connecting with the cannula at one end and the glass tube emerging from the stopper of the bottle at the other end are provided with a clamp for the control of the suction. A more convenient but expensive apparatus is that of Potain, in which a vacuum is made in the bottle by means of a pump. By means of a cock on the tube connecting cannula and bottle the negative pressure can be turned off. A low pressure is desirable.

Precautions in Tapping.—A finger of the left hand is placed firmly in the interspace to be pierced, and the trocar forced through in a vertical direction. An oblique insertion causes pain with every respiratory movement. The point should glide over the lower rib to avoid the intercostal artery, which lies underneath the upper rib. In his zeal to keep away from the artery the novice is likely to wound the highly sensitive periosteum of the under rib needlessly.

Before using the aspirating cannula on the patient its potency should be tested by sucking up sterile water. This is also an assurance that the bottle is properly exhausted. We once, by a wrong attachment of the pump, produced a positive instead of a negative pressure in the bottle. When the cock was turned air was forced into the pleural cavity, fortunately with no ill result.

Rate of Withdrawal.—The more slowly the exudate is withdrawn the safer is the operation. Slow drainage likewise gives the retracted lungs a better opportunity to expand without exciting congestion. Rapid evacuation of fluid tends to disturb both the pulmonary and systemic circulation. The arterial pressure tends to fall and the brain has an inadequate supply of blood. On these grounds we strongly recommend simple siphonage whenever the method succeeds in maintaining a flow; otherwise the minimum suction required for the purpose is advised.

Amount to Withdraw.—No definite routine can be carried out in determining the amount of fluid to be withdrawn. The question is best settled in each instance by the size of the effusion, by the ease of drainage, and by the condition of the patient during the procedure. Generally speaking, an effusion that escapes spontaneously indicates a good tonus of the lungs and a return of lungs and diaphragm to their normal position. Hence one may remove as much fluid as can be obtained by siphonage. On the other hand, an effusion requiring considerable aspirating force to induce its flow characterizes lung tissue less capable of reëxpansion; consequently less fluid should be removed. Fifteen hundred c.c. is a large quantity for evacuation. Beyond this amount great care is necessary. Certain clinicians have advocated the removal of only a few cubic centimeters, on the theory that absorption will take place, provided the excess pressure is relieved. Stintzing and Gerhardt both claimed some success with this treatment, but the results were not brilliant.[1] Certainly the evacuation of the greater proportion of fluid promotes absorption effectively, besides relieving the displaced organs.

---

[1] I have made it a habit for many years first to determine the character of the pleural fluid by withdrawing a few cubic centimeters with a hypodermic syringe. Unless there is need of hurry I wait for several days before withdrawing as much as

At any stage in drainage the onset of pain, severe cough, or faintness is a signal to stop the flow momentarily and, if the symptoms persist, to remove the trocar.

Untoward Symptoms During Thoracentesis.—The symptoms developing in the course of drawing off a pleural exudate demand the closest attention. They arise, in the main, from an inability of the viscera to adjust themselves to the sudden change of physical conditions. They therefore show a disposition to grow more pronounced or even to be a source of danger as the aspiration progresses unless the exciting cause, namely, the evacuation, is terminated.

Cough.—Cough may be expected to appear after two or three pints have been removed. It arises from excitation of the vagus nerves in the congested lung, or rarely from the direct irritation by the trocar of the same nerves in the pleura. By partly withdrawing the instrument the latter factor can be eliminated. Should the cough persist the trocar must be withdrawn. Occasionally cough is a troublesome complication before any considerable amount of fluid is obtained. It may be paroxysmal and uncontrollable. To proceed under such circumstances exposes the patient to risk of tearing the lung or of rupture. A preparatory injection of morphin (gr. ⅙-0.01 gm.) with atropin (gr. 1/100-0.0006 gm.) will render a second attempt more successful. Siphonage will be better tolerated than aspiration.

Faintness.—Faintness emanates from two sources, the one psychic, in which the cerebral circulation is directly disturbed by the excitement of the ordeal; the other reflex, in which the disturbance of the pulmonary circulation reacts on the cerebral. The former is relieved by the administration of aromatic spirits of ammonia (ʒss-2.0 gm.) in whisky ℥i-30.0 gm.) and need not interfere with the operation. The latter is not arrested by stimulants and demands a halt in the evacuation. In both types the pressure in the radial artery falls. In both the recumbent position soon restores the cerebral circulation.

Weak Pulse.—A weak pulse or a pulse of low pressure is not often encountered by the careful operator. The treatment mentioned for faintness is indicated. The serious forms will be described later.

Dyspnea.—Dyspnea is one of the exceptional incidents during aspiration and, as a rule, the result of paroxysmal coughing.

Pain.—Pain is ordinarily of no consequence when the trocar has once penetrated the parietal pleura. Its onset in the course of drainage may be explained by the pull of adhesions on the parietal or diaphragmatic pleura

---

I think is necessary. When this is done brilliant results may not be obtained, but a sufficient number of exudates are absorbed to make it worth while to try this method.—Editor.

brought about by the expansion of the lung. Or the lung may come in contact with the cannula and produce painful movements of the instrument in the wound. We once saw an epigastric angina suddenly develop with an abrupt rise in the blood-pressure (50 mm. Hg) as a consequence of a needle point catching in the pleura. For severe pain of every description the withdrawal of the instrument is the surest remedy.

Dangerous Symptoms and Their Treatment.—Tapping the chest is a safe operation when carried out by an experienced clinician. Delafield reports 200 cases aspirated in his hospital service without a death. Undoubtedly scores of patients with pleural effusion are sacrificed to ignorance or delay in receiving timely relief, while loss of life from operation is rare. Yet it cannot be said that puncture of the chest is as free from danger as puncture of the abdomen. Fortunately most of these dangers can be avoided by care and watchfulness.

Hemorrhage.—Hemorrhage from injury to the intercostal artery has been described. If the bleeding is profuse a ligature can be applied.

Pulmonary hemorrhage deserves more serious consideration, as it may be excessive and cannot be easily controlled. The liability to this complication is greatly enhanced by piercing a portion of consolidated lung, and especially a tuberculous focus. Unverricht emphasizes the prevalence of small aneurysms in tuberculous cavities, and attributes the bleeding to an acute engorgement of those vessels. Absolute quiet and morphin are the measures best adapted to check the loss of blood.

Infection.—Infection of the exudate with germs from the outside is not to be feared, provided the ordinary aseptic precautions are observed. Why accidental infection is not a more common occurrence has long excited the surprise of writers. True, a certain proportion of cases yield serous fluid at first and pus at a later tapping, but this change in the nature of the exudate may be spontaneous.

Pneumothorax.—Pneumothorax of small extent is frequent, because it is almost impossible to have all the joints of the aspirating instrument so tight that no air is sucked into the chest. Many look upon the entrance of air as a great menace, but in our own experience we have seen no harm result even from considerable quantities. Air driven into the chest under pressure soon gives rise to the same symptoms as are induced by an excessive accumulation of fluid. During the pleural drainage, however, the air drawn in preserves a relationship to the fluid withdrawn. The volume of air entering the chest seldom equals or exceeds the volume of effusion eliminated, so that no subjective symptoms arise. For days after drainage an area of tympanitic quality on percussion can be demonstrated, but after two weeks or so the air is absorbed. Pneumothorax resulting from a wound of the pleura has a more unfavorable prognosis, because the opening may persist and admit both air and infectious material from the bronchial tubes.

Subcutaneous Emphysema.—Subcutaneous emphysema is evidence that the trocar has pierced the lung and along the wound air has found its way to the subcutaneous tissues. The skin is elevated, and on pressure emits a crackling sound like the crushing of dry leaves. The air infiltration may be localized about the site of puncture or extend over a large portion of the body.

Edema of the Lungs.—This is at the same time the most formidable and insidious of the complications attending thoracentesis. The onset usually comes at the end of the drainage or within a few hours after. The attack may begin with cough with abundant sputum. The secretion is frothy, contains serum and blood, and may be expelled in enormous amount. The French have described the condition by the name of "expectoration albumineuse." The explanation of this phenomenon is ascribed to a great wave of hyperemia in a lung that has been long retracted and bound down by adhesions and unable suddenly to expand upon the release of the pressure of the exudate. The vessels in the compressed lung have lost their tone, and in this condition of paralysis the blood exudes into the tissues. Some authors favor the theory of Cohnheim that the vessels become morbidly permeable because of the impaired nutrition of the cells. The factors that seem to bring on pulmonary edema are rapid evacuation, the removal of very large quantities, and the use of strong aspiration. Rarely an attack comes on when a small effusion has been drained by siphonage.

The complication is sometimes fatal. Preventive measures are obvious, namely, to evacuate fluid slowly, to limit the amount, and to use little aspirating force. Uncontrollable coughing is sometimes a forerunner of edema.

For relief of the immediate symptoms morphin and atropin have given the most satisfaction. Adrenalin has been recommended, but as it actually causes pulmonary edema when given to rabbits it should be condemned.

Collapse and Sudden Death from Thoracentesis and Irrigation of Pleural Cavity.—In addition to the accidents already discussed there are cases of collapse and sudden death occurring either at the time of puncture, during aspiration or some hours after, or in connection with irrigation of the pleural cavity. Although the fatalities are few in comparison with the great number of successful operations, yet the condition deserves the fullest consideration in order to determine the causative factors and the means of combating them.

Leichtenstern in a critical review of the literature divides the cases into three groups:

1. Those in which death occurs during aspiration, coming on suddenly and without warning. He cites 4 cases of this character.

2. Those in which death follows aspiration, in a few minutes to several days. He finds 14 examples of this group.

3. Those in which a fatal issue attends irrigation of the pleural cavity. Five such cases he reports, some characterized by general or partial convulsions.

Auberne and Weill describe convulsive seizures accompanying irrigation of the pleural sac, sometimes followed by syncope and death, sometimes by recovery. The convulsive movements are said to be more pronounced on the side of pleurisy.

Wilson Fox analyzed the manner of death in 12 cases where, excluding instances of hemorrhage and edema, the patient succumbed within forty-eight hours of withdrawal of fluid. Seven died of syncope, 2 of asphyxia, 3 of convulsions. No organic lesions could be found in any case to account for the attack.

Russell contributes the most instructive discussion of the subject at our command, and furnishes an unusually rich experience. He reports 3 fatalities from exploratory puncture, all of them subjects of pneumonia. The syncope originates, in his opinion, in a direct injury to the vagus nerve fibers in the lung tissue, from which reflex impulses are carried to the heart. The same reflex mechanism is set in motion by an acute congestion of the lung in aspiration. He is not so convincing in his explanation of death from irrigation, for he considers the pleura is not concerned in the reflex, a conclusion entirely opposed to our own. In his animal experiments he found it impossible to excite such reflexes from the pleura.

Sears has recently published a fatality occurring fifteen hours after exploratory puncture. He also gives a full bibliography.

The subject has been studied by Lewis and the author, experimentally and clinically. In healthy animals we found very little circulatory disturbance from tapping the chest or from injecting and aspirating moderate quantities of oil. Nor was any untoward effect produced by irritation of the pulmonary pleura. Where, however, a pleurisy with effusion was produced by a previous injection in the pleural cavity of oil and turpentine the results were different.

In the first place, the forceful thrust of the trocar against the chest in its introduction often caused a transitory fall in arterial pressure which was attributed to direct mechanical displacement of the heart. In a dog the chest is narrow, so that this effect is exaggerated. But even with the broader thorax in man, with the heart already dislocated and embarrassed by effusion, a forceful puncture has a distinct inhibitory influence on the heart action, lasting usually a few seconds. This influence is greatest in the yielding chest of children.

Irritation of the inflamed visceral pleura by the trocar point or by chemicals in the process of irrigation in some cases caused a fall in arterial pressure that occasionally was fatal.

Clinically we observed a fatal syncope at the beginning of aspiration when only a few ounces had been withdrawn. In a number of cases of pleurisy during evacuation we have found that scratching the pleura with a needle point or cannula is followed by a fall in arterial pressure which if protracted leads to faintness.

Similar evidences of circulatory disturbance were noted in some instances in which aspiration was rapid or the amount of fluid withdrawn excessive. The salient features of these attacks of syncope are:

1. In fatal cases no anatomical lesion can be found to explain the accident.
2. The symptoms may develop suddenly (*a*) at the moment of puncture as a result of direct cardiac inhibition; (*b*) at any time during aspiration, but especially toward the end. The risk is increased by using strong suction and by withdrawing too much fluid. The chief factor is the irritation of the pleural nerves by the trocar or the pulmonary nerves by the congestion of the lungs, (*c*) immediately after or within the forty-eight hours following aspiration due to the congestion of the lungs, (*d*) during irrigation of the pleural cavity with antiseptic solutions, for example, formalin, iodin, etc. The chemicals irritate the pleural nerves and excite reflex circulatory disturbances.

The treatment most to be emphasized is preventive. The instrument selected for thoracentesis should be sharp, so that no undue force is demanded to penetrate the chest wall. At the same time, it should not irritate the visceral pleura after introduction any more than is absolutely necessary. The trocar meets these requirements better than the needle. The *trocar* should not be inserted to a greater depth than is needed to obtain fluid. Care should be taken during the drainage of an empyema to *avoid a long projection of the drainage tube* inside the cavity. Swabbing the pleural surface is attended with danger. Finally, *siphonage* or slow aspiration is desirable. The emergency treatment of collapse is, first, to place the *head low,* as in shock, and to *elevate the foot* of the bed. *Artificial respiration* must be instituted if the respirations have ceased. Few drugs are of any value.

Atropin is useful when the heart is slow and irregular, but of no aid when the pulse is weak and rapid. *Adrenalin,* 20 minims (1.3 gm.) of a 1:1,000 solution, diluted in several cubic centimeters of salt solution and injected in a vein, is the most efficient remedy. If the pulse is gone it is better to inject the drug directly into the right ventricle of the heart.

Intrapleural Injections to Aid Absorption.—In 1895 Hamburger completed his investigations which proved that fluid exudates in serous cavities must become isotonic with the blood-serum before absorption can take place. Stimulated by this idea Lewaschew and others practiced repeated tapping of old exudates, and followed this by injections of physiological *salt solution.* By equalizing the molecular density of the fluid with that of the blood they hoped to favor absorption, but had little success. Achard was one of the first to introduce *sterile air* in the thorax to stimulate absorption. More recently he has used ordinary air for the purpose of replacing the fluid and securing more complete evacuation of the exudate. By forcing in air as the fluid escapes he claims that even three or four liters can be removed with safety.

The author finds it expedient to allow air to be sucked in through the cannula at intervals during the process of evacuation, if much force is necessary to draw out the effusion. The danger of air infection is no greater than when the abdominal cavity is opened in tuberculous peritonitis.

Oxygen gas is praised by A. Schmidt as the superior of air or of nitrogen in promoting absorption, as the gas itself disappears from the chest.

Thiosinamin has been advocated by Schnütgen and others for the purpose of bringing about a digestive solution of fibrinous exudate and of preventing its absorption. One part of thiosinamin is diluted with 5 parts each of glycerin and water. Two minims of this preparation are injected into the muscles twice a week, and the dosage gradually increased to 1 c.c. Fibrinous adhesions are thus prevented, but reliable conclusions cannot be drawn from a small number of cases. On theoretical grounds a digestive substance capable of dissolving the fibrin obstructing the lymph spaces would be a great aid to absorption.

Adrenalin chlorid has the theoretical merit of constricting the blood-vessels and diminishing the secretion of the pleural cells. Plant and Steele first evacuate the serous collection as completely as possible and then inject through the cannula 1 dram (4 c.c.) of adrenalin chlorid (1:1,000) diluted to 1 ounce of sterile water.

Ewart advocates the injection of 10 minims (0.6 c.c.) of the above solution as a preliminary to thoracentesis. The result is often "a spontaneous reabsorption without any need for operation." Our own experience with adrenalin injections has not been so encouraging.

Autoserotherapy.—Gilbert in 1894 first suggested the aspiration of a few cubic centimeters of serous fluid and its reinjection under the skin. He noticed that subsequently absorption was materially accelerated, although he did not venture any explanation of the phenomenon. Fede commended the treatment, but had no theory of its working, except a belief in some stimulating effect from repeated mechanical irritation of the pleura with the trocar. Tschigaeff tried this method in 9 cases, and found that always after the injection the effusion ceased to increase and absorption began. The duration of a serous pleurisy on an average was shortened to two weeks. The earlier the injection the prompter was the recovery.

Schnütgen was also favorably impressed by his experience with the method.

Zimmerman considers the results of autoserotherapy are to be attributed to a reactive leukocytosis that exerts an absorptive influence on the exudate.[2]

Chronic Serous Effusion.—There is a group of cases in which the exudate reaccumulates persistently in spite of all therapeutic measures, including repeated thoracentesis. Effusions accompanying endothelial tumors of the pleura exhibit such a tendency, and call for purely symptomatic measures. Other cases of unknown origin have been treated by thoracotomy with the hope of bringing the inflammatory reaction to an end. But, in the words of Forchheimer, "it is simply a matter of chance whether or not incision and drainage will correct the condition which produces the chronic pleurisy."

After Treatment.—In a certain proportion of cases repetition of the aspiration may be necessary once or many times. A reaccumulation of the effusion in a quantity to reach the level of the third rib is sufficient indication for another evacuation. A period of a week or more usually intervenes between the first and second tapping.

When the fluid has been entirely absorbed, or nearly so, and the fever has subsided, the patient should be encouraged to take *moderate exercise* and practice *deep breathing*. *Walking* slowly out-of-doors with shoulders thrown back and taking full inspirations admirably fulfil these requirements. Later on the reëxpansion of the lung can be further assisted by the use of special apparatus. A simple device consists of two large Wolff bottles connected by tubing, the one empty, the other containing water. From the latter a second tubing emerges, fitted with a mouthpiece. The patient blows the water from one bottle into the other, and this exercise he repeats a prescribed number of times every day.

Diet.—The diet should be nutritious and liberal. To recover a pronounced loss of weight, milk, buttermilk, and eggs should be given between meals.

Hydrotherapy.—Hydrotherapy is an important aid in convalescence. A daily bath in cool water stimulates the circulation and improves the appetite.

Outdoor Life.—An out-of-door life should be enjoined for at least three months to a year, depending on the severity of the attack. One must ever keep in mind the danger of an extension of tuberculous pleurisy or of an invasion of tubercle bacilli upon the site of a recently healed pleurisy of some other origin. An extended sojourn by the sea, in the mountains, or in the country is highly important and should be insisted upon whenever possible.

---

[2] I have tried autoserotherapy in a number of patients with pleural effusion and have never been able to convince myself that it had any effect whatever.—Editor.

Chylothorax.—By the term chylothorax we understand the presence within the pleural cavity of any fluid which has a milky appearance either from the presence of a true chyle or from chyliform exudate. The presence of *true chyle* depends on some destruction or injury to the thoracic duct, whereby its contents are emptied into the pleural cavity. Traumatism, tumors pressing from without, new growths in the duct, destruction of the left subclavian vein, and parasites are the etiological factors in their order of frequency. The *chyliform* exudate contains not chyle, but fatty globules derived from degeneration of the pleural epithelium and pus cells. Carcinoma and tuberculosis are usually associated with this form.

Treatment.—The treatment of chylothorax is dependent on the cause of the complication and on the symptoms produced. A traumatic injury to the duct sometimes heals spontaneously. In cancerous cases palliative measures only are possible. Chylothorax of tuberculous origin may be evacuated with some hope of recovery. Operative measures are rarely successful.

Hemothorax.—Pleural transudates of cardiac or renal origin always contain red corpuscles, but seldom enough to impart a deep tinge to the fluids.

Hemorrhagic Pleurisy.—Hemorrhagic pleurisy is characterized by a larger amount of blood mixed with the serum. Most of these exudates originate in a cancerous or tuberculous inflammation of the pleura, occasionally in a pleurisy due to pneumococcus infection.

Remedial measures for a hemorrhagic pleurisy are essentially the same as for a serous, except that one should be more conservative in the amount of exudate withdrawn in the former type. In these cases the effusion is often under very little pressure and must be pumped out. As the lung may be adherent and may not expand, a strong negative pressure is created in the chest which favors congestion and rapid refilling. Such an undesired result may be obviated by allowing air to be sucked in through the cannula until no more will enter. Air should, however, not be forced in.

Hemorrhage may result from rupture of aneurysm or the erosion of any intrathoracic arteries or veins. Rarely bleeding from a tuberculous cavity enters the pleural space.

Traumatic Form.—The traumatic form of hemothorax is encountered in fracture of the ribs, contusions of the chest wall, or injury to internal vessels by penetrating gunshot wounds. The treatment is surgical. Parietal bleeding may be stopped by ligature. Hemorrhage from the pulmonary vessels may gradually cease under absolute rest and the influence of opiates. Unfortunately, little can be done if the larger vessels are involved. Laying open the chest to find the bleeding point induces complete pneumothorax and endangers life. There is hope that with the negative pressure apparatus of Sauerbruch or the positive pressure device of Brauer pneumothorax can be avoided and the operation be made successful. By keeping the lung inflated the bleeding vessel can be more easily located. Moreover, the hemorrhage itself is favorably influenced, for it has been found by Sauerbruch that less blood flows through an inflated than a collapsed lung.

Pneumothorax.—The harmlessness of a small or moderate quantity of air in the chest is shown by the fact that air has been many times injected into the cavity for therapeutic purposes without embarrassing respiration or circulation. Most of the instances of pneumothorax coming to the attention of the internist are closed.

An open pneumothorax allowing free entrance and egress of air is more often seen by the surgeon as a result of chest wounds or thoracotomy. The danger of such free openings, according to the investigations of Graham, depends upon the ratio of the size of the opening to the vital capacity of the lungs. If the vital lung capacity is high, a large opening may be compatible with life; if, however, the vital capacity is low, only a small opening can be tolerated.

Accidental Pneumothorax during Thoracentesis.—We have seen many instances of circumscribed or partial pneumothorax following thoracentesis, and it is exceptional that any symptoms arise therefrom. The air may enter through a leaky valve or through the cannula while clearing out an obstruction with a stilet. Such an accident need cause no apprehension and calls for no treatment. When, however, by making the wrong connection with the Potain pump, air is forced into the chest, alarming symptoms may develop. The patient gasps for breath, respirations are shallow, the pulse grows feeble, and consciousness may be lost. Under such circumstances immediate release of the air through the disconnected trocar or by aspiration is required to save the patient's life. In the presence of fluid, air has been purposely allowed to enter the chest during inspiration in order to aid the spontaneous outflow of serum. So long as the column of air introduced does not exceed the volume of fluid evacuated, no harm results.

Pneumothorax Acutissimus.—The "suffocative" pneumothorax, or pneumothorax acutissimus, is the most alarming form of this disease, and calls for prompt action on the part of the physician. It is caused by a "valvular" opening in the lung that permits the entrance of air with inspiration, but prevents its escape with expiration. Consequently air collects in the cavity until it will hold no more, and the patient's struggle for breath steadily increases.

At the onset *morphin* may be employed to arrest the pain and dyspnea.

Cardiovascular stimulants, such as camphor and caffein, are helpful in combating signs of failure on the part of the circulation.

As soon as the diagnosis of pneumothorax can be verified by physical examination *thoracentesis* is indicated. It is better to allow air to escape through the cannula than to use suction; for aspiration of the gas tends to re-open the lung fistula. Puncture may have to be repeated. *Continuous drainage,* either by leaving the cannula in place or by insertion of a rubber tube in a pleurotomy wound, has been recommended and successfully practiced. Béclère advised attaching to the trocar a rubber tube twenty inches long, the distal end of which is immersed in a test tube of water. This prevents the inspiration of air. By this procedure the intrapleural pressure does not exceed that of the atmospheric air, and healing of the lung perforation is favored.

Traumatic Pneumothorax.—The treatment is most cases of traumatic pneumothorax is symptomatic. A moderate amount of air is absorbed by the pleura and may cause little inconvenience. The chief danger lies in the *infection* of the serous membranes. Traumatic rupture of the lung is less likely to be followed by a pleuritis than perforation of the chest wall. If the wound seems clean, it should be disinfected and sealed, thus converting an open into a closed pneumothorax. Whenever it is evident that the pleura is infected a drainage tube should be inserted.

Spontaneous Pneumothorax.—The amount of gas produced by organisms in the pleural cavity seldom is sufficient to excite symptoms. The treatment of the associated empyema is the important consideration.

Tuberculous Pneumothorax.—The entrance of air into the pleural cavity is not always a misfortune to the subject of pulmonary tuberculosis. Oftentimes the resulting compression of the lung leads to healing (Spengler). It is unwise, therefore, to withdraw the air by puncture unless urgent dyspnea and cyanosis develop, or unless there also exists an excessive serous or purulent exudate.

Pneumothorax with Serous Effusion.—It has been emphasized that the air introduced into the pleural cavity generally carries with it organisms that excite a pleuritis with a fluid exudate. Hence, we are confronted with the necessity of treating two distinct diseases. Emerson believes that, in general, the air may be disregarded and the effusion treated. Thoracentesis is not to be practiced as a routine measure. The size of the accumulation, the degree of dyspnea, and the rate of absorption must be the deciding factors. Whenever puncture is performed, siphonage is preferable to aspiration.

Pneumothorax with Empyema.—Perforation of an empyema into the bronchi rarely leads to pneumothorax. Likewise, external spontaneous perforation (*empyema necessitatis*) creates such a small fistulous opening in the chest wall that air seldom enters the pleural cavity, as the pus escapes slowly and gives the lungs time to expand. On the other hand, the surgical operation of pleurotomy drains off the pus so rapidly that the lung expansion cannot keep pace, and air is sucked in freely. In fact, a certain degree of pneumothorax is desirable, since it promotes the evacuation of the cavity.

Treatment.—The treatment considers less the pneumothorax than the empyema. The air may often be ignored but the pus must be drained by a free incision. In all cases, of whatever nature, prompt surgical measures will give the patient his only chance of recovery.

Putrefactive Exudates.—Putrefactive exudates are of even greater menace than purulent, and call for the same radical treatment. Simple *puncture,* except for diagnostic purposes, has little to recommend it, and gives only temporary relief.

Supportive Measures.—Supportive measures are needed whether the treatment is expectant or operative.

A liberal, nutritious *diet* tends to build up the individual's strength and resistance. *Whisky* and wine, alone or in an eggnog, are serviceable. One of the best stimulants for the circulation is *coffee.*

As previously stated, *morphin* should be freely employed in pneumothorax because of its sedative influence on the respirations and its relief of pain. It should be given hypodermatically.

Artificial Pneumothorax for Therapeutic Purposes.—In the majority of pleural effusions evacuation of the fluid is followed by expansion of the lung and obliteration of the space previously occupied by the fluid. But in many old serous effusions and in most chronic hemorrhagic effusions of tuberculous or cancerous origin, the lung is so bound down by adhesions that it fails to expand, thereby leaving an empty space under high negative pressure. As a consequence the exudate quickly collects and the inflammation is often aggravated. In these cases the injection of nitrogen gas or air into the cavity is of great service.

Weil reports that of 86 cases of serofibrinous pleurisy treated by puncture alone, 84 per cent showed marked secondary trouble a few weeks or months later, while of 50 cases in which air was introduced after the evacuation of fluid, 82 per cent recovered without adhesions. The air will not introduce any bacteria if filtered through a glass cannula packed with sterile cotton. When fluid is under little pressure and fails to flow, or when much suction is required, the introduction of a small amount of air will frequently establish a flow.

TUMORS OF THE PLEURA

A variety of tumor growths are encountered in the pleural membranes, including carcinoma, sarcoma, lipoma, enchondroma, fibroma, as well as the echinococcus cysts. Most of these neoplasms are *secondary* to growths in neighboring organs, more especially in the mammary gland, the stomach, the liver, and the mediastinal lymph glands. Fränkel has described the endothelial carcinoma of the pleura, which he looks upon as primary.

Treatment.—Palliative measures only can be carried out. Withdrawal of the exudate by puncture is indicated if dyspnea is present. The operation should be repeated as often as expedient. In the presence of a rich hemorrhagic effusion greater caution is necessary. The patient is entitled to the benefit of opiates if pain is pronounced.

Echinococcus Cysts.—Echinococcus cysts have been successfully drained with the trocar, but, as Lord points out, the procedure has been attended with too high a mortality to be recommended. The cyst should be removed without rupture, and to accomplish this costectomy is the operation of choice.

REFERENCES

Achard. Sur l'Insufflation d'Air stérilisé dans les Epanchements Pleureaux, Bull. et mém. Soc. méd. d. hôp. de Paris, 1903, xx, 3, 430.

Aron. Die Mechanik und Therapie des Pneumothorax, 1902.

Auberne. Rev. de méd. et de chir., Feb., 1879.

Auerbach. Pleuritis in ihrer Bedeutung für Diagnose und Therapie der Abdominalerkrankungen, München. med. Wchnschr., March 7, 449, 1905.

Bard. Experimental and Clinical Researches on the Intrapleural Pressure in Pneumothorax, Rev. de méd., xxi, 1901.

Béclère. Traitement du Pneumothorax. Rev. gén. de Clin. et de Thérap., xi, 561, 1897.

Bergeat. Thorax Resection in Chronic Empyema, München. med. Wchnschr., 26, 1398, 1908.

Billings, F. T. Purulent Pleuritis—Treatment, Journ. Am. Med. Ass., lii, 266, 1909.

Blaker. Observations on Empyemata in Children, Brit. Med. Journ., i, 1200, 1903.

Bohland. Uber die Behandlung des Empyems mittelst der Heberdrainage, Deutsche med. Wchnschr., 48, 1893.

Bouveret. Empyema, Paris, 1888.

Bowditch, H. I. On Pleuritic Effusions and the Necessity of Paracentesis for Their Removal, Am. Journ. Med. Sc., xxiii, 320, 1852.

Bowditch, V. Y. Quoted by Musser in Chapter on Diseases of the Pleura, Trans. of Nothnagel's Encycl. Pract. Med., 820, 1903.

Braun. Empyema, Deutsche med. Wchnschr., 14, 1906.

Brewer. Surgery of the Thorax, Keen's Surgery, iii, 532, 1908.

Bunting. The Etiology of Serofibrinous Pleurisy with Reference to

Cystological Diagnosis, Johns Hopkins Hosp. Bull., xiv, 185, 1903.

Capps. Some Observations on the Effect on Blood Pressure of the Withdrawal of Fluid from the Thorax and Abdomen, Journ. Am. Med. Ass., xlviii, 22, 1907.

——— An Experimental Study of the Pain Sense in the Pleural Membranes, Arch. Int. Med., vii, 717, 1911.

——— and Lewis. Observations Upon Certain Blood Pressure Lowering Reflexes That Arise from Irritation of the Inflamed Pleura, Am. Journ. Med. Sc., cxxxiv, 868, 1907.

Capps and Lewis. Blood Pressure Lowering Reflexes from Irrigation of Chest in Empyema, Arch. Int. Med., ii, 166, 1908.

Carrière. Traité des Maladies de l'Appareil Respiratoire, 2d ed., 596, Paris, 1908.

Councilman, Mallory, and Pearce. A Study of the Bacteriology and Pathology of 220 Fatal Cases of Diphtheria, Journ. Bost. Soc. Med. Sc., v, 159, 1900.

Cushny. Pharmacology and Therapeutics, 4th ed., 5 London, 1906.

Dayton. Accidents and Deaths from Exploratory Puncture of the Pleura, Surg., Gynec. & Obst., xiii, 6, 607, 1911.

Delafield. The Treatment of Pleurisy with Effusion, Am. Journ. Med. Sc., cxxiv, 6, 939, 1902.

Dybkowsky. Quoted by Garland.

Elsberg. Pneumothorax and Posture, Journ. Exper. Med., xi, 444, 1909.

Emerson. Pneumothorax, an Historical, Clinical, and Experimental Study, Johns Hopkins Hosp. Rep., xl, 1903. This work is perhaps the most exhaustive historical and clinical treatise ever written on the subject. The monograph also contains much original matter on various phases of the mechanics of pneumothorax.

Emerson, H. Intra-abdominal Pressures, Arch. Int. Med., vii, 754, 1911.

Ewart. Pleuritic Effusion Treated with Adrenalin by the Pulmonary Injection Method, Brit. Med. Journ., April 28, 973, 1906.

Fede, F. L'Autoseroterapia o Cura Gilbert nelle pleuriti sierofibrinose, Riforma med., xxii, 1319, 1906.

Feidler. Zur Etiologie der Pleuritis, Jahresb. d. Gesellschaf. f. Nat. u. Heil., Dresden, 1891.

Finley, F. G. Pneumothorax from Gas-producing Bacteria, Montreal Med. Journ., xxviii, 759, 1899.

Fleiner, W. Uber die Resorption corpusculäres Elements durch Lungen und Pleura, Virchow's Arch. f. path. Anat., etc., cxii, 2, 282, 1888.

Forchheimer, F. The Treatment of Pleurisy with Effusion, Journ. Am. Med. Ass., xlviii, 28, 1907.

Forgues. Arch. d. méd. et pharmacie mil. Par., xxiv, 1894.

Fox, W. Diseases of Lungs and Pleura, London, 1891.

Fränkel, A. Uber primären Endothelkrebs der Pleura, Berl. klin. Wchnschr., 21, 497, 1892.

——— Spezielle Pathologie u. Therapie der Lungenkrankheiten, Berlin und Wien, 1904.

Fräntzel. Krankheiten der Pleura, Leipzig, v. Ziemssen's Handbuch der speciellen Path. u. Therapie, iv, 2d ed.

Galliard. Suffocative Pneumothorax Treated by Pleurotomy, Bull. et Mém. d. Soc. Méd. d. Hôp. de Paris, xviii, 6, 1901.

Garland. Pneumodynamics, New York, 1878.

Gerhardt. Die Pleurakrankungen, Deutsche Chirurgie, iv, 1892.

Grawitz, E. Zur Physiologie und Pathologie der Pleura, Berl. klin. Wchnschr., xliii, 29, 621, 1897.

Hamburger. Uber Resorption aus der Peritonealhöhle, Arch. f. Anat. u. Physiol. Abtheil. 36, 1896.

Hebb. Primary Cancer of the Pleura, Tr., Path. Soc., xliv, 5, 1893.

Hill, Leonard. The Relative Influence of Heat and Chemical Impurity of Close Air, Journ. Physiol., xli, 3, 1910.

Hoover. Some Observations on the Physical Signs of Pneumothorax and Its Treatment, Cleveland Journ. Med., Feb., 1898.

Hopkins. The Operative Treatment of Pneumothorax and Pyothorax, Ann. Surg., xxvii, 216, 1898.

Janeway, E. G. Hemiplegia as the Result of the Use of Peroxid of Hydrogen in a Sacculated Empyema, Am. Journ. Med. Sc., cxvi, 420, 1898.

Karsner and Swanbeck, Journ. Med. Res., 1920, xlii, 91.

Laënnec, R. T. H. Treatise on Diseases of the Chest, First American Edition, Philadelphia, 1823.

Léger. Contribution à l'Etude du Traitement du Pneumothorax tuberculeux, specialement des Pleurésies, Thèse, Paris, 1898.

Leichtenstern, O. Plötzliche Todesfälle bei pleuritischen Exsudaten, Deutsches Arch. f. klin. Med., xxv, 325, 1880.

Lewaschew. Wratsch, xlii, xliii, 1895.

Lichtheim. Die Störungen des Lungenkreislaufes und ihr Einfluss auf den Blutdruck, Berlin, 1876.

Lord, F. T. Chapter on Pleuritis, Osler's System of Modern Medicine, iii, 1907.

May and Gebhard. Pneumothorax durch Gasbildende Bacterien, Deutsches Arch. f. klin. Med., lxi, 1898.

Maydl. Uber Echinococcus der Pleura, Wien, 1891.

Meininger. Quoted from Yeo's Manual of Med. Treatment, 14th ed., i, 650, 1906.

Miller, J. L. Transudates and Exudates, Am. Med., viii, 835, 1904.

Murphy, J. B. Surgery of the Pleura, N. Y. Med. Journ., lxxxix, 7, 356, 1909.

Musser. Osler's System of Modern Medicine, Philadelphia, ii, 564, 1907.

Neelsen. Untersuchungen ueber den Endothelkrebs, Deutsches Arch. f. klin. Med., xxxi, 375, 1882.

Netter. Recherches Experiment. sur l'Etiologie des Pleurésies, Abstract from Centralbl. f. klin. Med., 784, 1891.

——— De la Pleurésie Purulente Metapneumonique et de la Pleurésie Purulente Pneumococcique Primitive, Bull. et mém. d. Soc. méd. d. hôp. d. Paris, series 3, vi, 13, 1889.

Opie. Transformation of Serofibrinous into Purulent Pleurisy, Journ. Exper. Med., ix, 414, 1907.

Ortner, N. Zur Klinik des Pyothorax u. des Pyopneumothorax, Berl. med. Klin., 38, 1422, 1909.

Osler. Spontaneous Pneumothorax, Maryland Med. Journ., xxxviii, 1898, 461.

Otto. Uber den pleuritischen Schmerz und seine Behandlung durch partielle Compression des Thorax, Berl. klin. Wchnschr., 39, 852, 1889.

Pflügge and Coworkers. Ztschr. f. Hyg. u. Infections-krankh., lix, 1905.

Plant and Steele. Treatment of Serous Effusions by Adrenalin Chlorid, Brit. Med. Journ., July 15, 125, 1905.

Quincke. Uber den Druck in Transudaten, Deutsches Arch. f. klin. Med., xxi, 1878.

Ramström. Merkel's and Bonnet's Anat., xxx, 92, 1906.

Reineboth. Experimentelle Studien über die Wiederausdehrung der Lunge bei offener Brusthöhle, ins besondere nach Operation des Empyems, Deutsches Arch. f. klin. Med., lviii, 178, 1897.

Riesman. Albuminous Expectoration following Thoracentsis, Am. Journ. Med. Sc., cxxiii, 4, 620, 1902.

Romberg and Pässler. Untersuchungen über die Pathologie der Kreislauf-störung bei acuten Infectionskrankheiten, Deutsches Arch. f. klin. Med., lxix, 652, 1899.

Rosenbach. Virchow's Arch., cv, 215, 1886.

——— Diseases of the Pleura, Am. Transl. of Nothnagel's Spec. Pathol. und Therap., Philadelphia, 848, 1903.

Russell, A. E. Death Occurring During or After Exploratory Puncture of Lung, St. Thomas' Hosp. Rep., xxviii, 465, 1899.

Saugman. Uber die Anwendung des künstlichen Pneumothorax in der Behandlung der Lungentuberkulose, Ztschr. f. Tuberk., xii, 1, 20, 1908.

Schmidt, A. Pain, Its Causation and Diagnostic Significance in Internal Diseases, transl. by Vogel and Zinsser, Philadelphia., 286, 1908.

Schmidt, A. Intrapleurale Injectionen zu therapeutischen Zwecken, Verhandl. d. Cong. f. innere. Med., 380, 1906.

Schnütgen. Uber pleuritische Schwarte und ihre Behandlung mit Fibrolysin, Berl. klin. Wchnschr., li, 2269, 1908.

Schnütgen. Die Autoserotherapie bei serösfibrinöser Pleuritis, xlvi, 3, 97, 1909.

Schreiber. Uber Pleural und Peritonealdruck unter patholog. Verhältnissen, Deutches Arch. f. klin. Med., xxxiii, 5, 485, 1883.

Sears. Accidents Following Thoracentesis, Tr. Ass. Am. Physicians, xxi, 177, 1906.

Sewall. Progr. Med., Sept. 69, 1909.

Spengler, L. Zur Chirurgie des Pneumothorax, Beitr. z. klin. Chir., xlix, 68, 1906.

Stintzing. Penzoldt u. Stintzing's Handbuch der Therapie der inner. Krankheiten, 3d ed., iii, 453, 1902.

Traube. Zur Lehre vom pleuritischen Exudat, Berl. klin. Wchnschr., 7, 448, 1872.

Trousseau. Clin. Med. transl. from 3d ed., 1, 556, 1882.

Tschigaeff. Auto-serotherapie, Klin. therap. Wchnschr., x, 12, 324, 1903.

Unverricht. Ebstein-Schwalbe, Handbuch der praktischen Medizin, 2d ed., i, 405, 378, 417, 1905.

Weil. Arch. f. klin. Med., xxv, Dec. 10, 1879.

——— Bull. de l'acad. de med., 1919, lxxxi, 846.

Weill. De la mort subite dans la pleurésie, Rev. dé méd., Jan., 1887.

West, B. The Prognosis of Pneumothorax with Some Statistics as to Mortality, Lancet, i, 1264, 1897.

West, S. Diseases of Organs of Respiration, 2d ed., London, 1909.

Widal and Ravaut. Applications Clin. de l'Etude Histologique des Epanchements Serofibrineux de la Plèvre, Compt. rend. Soc. de biol., Paris, lii, 651, 1900.

——— Permeabilité Pleurale du Salicylate de Soude, Bull. et mém. Soc. méd. d. hôp. de Paris, Series 3, xvii, 823, 1900.

Wintrich, M. A. Krankheiten der Respirationorgane, Virchow's Handb. der spec. Pathol. u. Therap., i, 1834.

Zimmermann. Zur Autoserotherapie der exudativen Pleuritis, St. Petersb. med. Wchnschr., xxxiv, 461, 1909.

CHAPTER VII

DISEASES OF THE MEDIASTINUM

George Blumer

MEDIASTINAL TUMORS

The treatment of mediastinal tumors is by choice curative, but, unfortunately, in many instances there is no hope of effecting a cure, and only palliative treatment is available. This is partly due to the fact that the symptoms of mediastinal tumor are due to the pressure of the neoplasm upon the important structures which lie in the mediastinum, so that, by the time these symptoms appear, the growth has already reached a considerable size in most instances. Another factor of importance is the very close relationship of malignant tumors to the important structures within the chest, which renders complete removal of the growth impossible. A third difficulty—the inaccessibility of such growths to surgical interference—has been very largely removed by the introduction of operations under positive or negative air pressure.

Curative Treatment.—The curative treatment of mediastinal tumors depends partly upon the nature and partly upon the situation of the growth. In the case of non-malignant tumors, such as adenomata of misplaced thyroid tissue, enlargement of the thymus gland, lipomata, or fibromata, surgical intervention is always indicated when pressure symptoms have appeared, and should not be delayed until the general condition of the patient is so poor that the resistance to the effects of hemorrhage, or of infection, is diminished. When, as occasionally happens, such tumors present in the neck, above the sternum or the clavicles, their removal is a comparatively simple matter, as it does not necessitate the resection of ribs or of portions of the sternum. Furthermore, such tumors are usually encapsulated, and the danger of opening the pleural cavity or injuring the great vessels is comparatively slight. In the case of the thymus the gland is usually easily removed on splitting the capsule. Under such circumstances ordinary surgical principles, such as hold in the removal of any non-malignant neoplasm, should be followed.

When mediastinal tumors, even non-malignant ones, are situated so that they cannot be reached from the neck, the problem of their removal becomes a much more complicated one. Nevertheless, surgical interference offers the only hope for recovery even in non-malignant growths, for, unless they are removed, death will result from mechanical pressure on the vital structures within the chest. In malignant growths surgical intervention is practically valueless so far as complete removal is concerned, for the great vessels and nerves, the esophagus, the bronchi, or even the heart itself are so intimately involved in the neoplasm, in most instances, that complete removal is an impossibility. Growths originating from the posterior surface of the sternum or the ribs, and growing into the mediastinum, have occasionally been successfully extirpated, but such growths can hardly be regarded as mediastinal neoplasms in the strict sense. Burnham states that no malignant mediastinal tumor has been cured by surgical means.

The non-malignant mediastinal growths which have been most successfully removed by the knife are the dermoid cysts or teratomata, and this is because it has been possible to diagnose them in a fair percentage of cases. In practically all instances their removal has necessitated the resection of a certain number of ribs, and sometimes also of a portion of the sternum. In a general way it may be stated that any operation for the removal of a mediastinal dermoid should be conducted within reach of either positive or negative pressure, obtained either by means of the Sauerbruch cabinet or by some of the simpler methods, such as that devised by Flint. The use of the methods of lung surgery in these cases may be necessary, because it is often impossible to avoid the production of a pneumothorax during the course of the operation, and this has, in some instances, resulted in a fatal outcome. The second point to be borne in mind is that resection of a sufficient number of ribs and, if necessary, of a sufficient quantity of sternal tissue, to give free access to the tumor, must be accomplished. In recent years the transsternal route has increased in favor with surgeons. Most surgeons who have operated on such tumors have laid stress upon the fact that there is usually a considerable formation of adhesions, although von Eiselsberg claims that their importance has been exaggerated. The fact remains that, of 15 cases of operation collected by von Eiselsberg in 1903, the removal of the tumor was complete in only 3, and in 2 of these 3 instances it was very small. While complete removal is certainly desirable, and is strongly urged by surgeons, it is obviously not always practicable. When impracticable, as much as possible should be removed, and the interior of the remaining portion of the cyst should be treated so as to encourage the formation of granulation tissue, which will subsequently cicatrize and lead to the obliteration of the sac. For this purpose some surgeons recommend curettage of the lining membrane, while others recommend that it be painted with caustic solutions. Whatever method of treatment is adopted, there is always danger when portions of the cyst wall are left behind that secondary hemorrhage may occur, or that local infection will extend to the other structures of the mediastinum, or will cause general sepsis. In order to avoid a pneumothorax, many surgeons recommend that, when a portion of the cyst is left, this should be sufficient to allow the edges to be drawn into the external wound and to be sutured to the skin. This procedure not only tends to prevent pneumothorax, but also makes more difficult an extension of infection, and is of great advantage in the treatment of secondary hemorrhage, should this occur.

The success of operative treatment in cases of dermoid cyst of the mediastinum has been very encouraging. Of 20 operative cases collected by R. S. Morris in 1905, 14 (or 70 per cent) survived and were cured, or else greatly benefited, by the operation. There is no reason why other non-malignant tumors of the mediastinum should not be as successfully removed. As a matter of fact, the difficulty lies not so much in the removal as in the diagnosis. Non-malignant tumors, other than dermoids, are usually accidental, postmortem finds.

There have been occasional cases of mediastinal dermoid which have yielded to measures less radical than extirpation, and this has been especially true of cases where the cystic character of the growth has been the predominant one. Kidd has reported such a case where the tumor presented in the epigastrium and where complete recovery followed simple incision and drainage. Such cases are exceedingly rare, but this method should be considered where the situation of the neoplasm allows it to be carried out.

Palliative Treatment.—The palliative treatment of mediastinal tumors may have for its object an effect upon the tumor itself, the relief of the intrathoracic pressure by surgical means, or the relief of symptoms.

There is a type of malignant mediastinal neoplasm usually described in the literature as lymphosarcoma which seems amenable to amelioration as a result of the action of a variety of agencies. These may be either physical, chemical, or infectious, and the action of all of them is presumably dependent upon the fact that the cells of certain varieties of sarcoma are extremely susceptible to their destructive power.

In some instances a temporary diminution in the size of mediastinal growths of this type has been observed to follow an infection, such as erysipelas, and there is little doubt that the cases of malignant neoplasm which have been favorably influenced by Coley's mixed toxins of the streptococcus and prodigiosus have been so affected by a destructive effect of the toxins on the tumor cells.

In a certain number of cases, arsenic is stated by Ruff to be a medicament acting in this way, and operative procedures, such as exploratory incisions with mere exposure of the tumor, have sometimes had a similar result.

The remedy, however, which may frequently be relied upon in certain types of mediastinal growth to produce a very definite and decided effect is the X-ray. There are now in the literature a considerable number of cases of malignant mediastinal growth in which temporary amelioration has followed exposure to the X-ray. The method of employing this agent is not always accurately stated by the reporters, but apparently results have been obtained both with long exposures at comparatively infrequent intervals and with short exposures at frequent intervals. On account of the danger of X-ray burns, the latter method would seem to be the preferable one. Pfahler recommends a high vacuum tube with one ampere of current and a focal distance of 15 to 18 inches. He exposes the patient from twenty to thirty minutes six days in the week, and in some of his reported cases has kept this up for five months. Kienboeck exposes an area about 12 cm. in diameter for five or six minutes with a hard or moderately hard tube at a focal distance of 12 cm. He suggests mapping out the chest wall over the tumor into areas of 12 cm. in diameter, and exposing a new area at each sitting. After the front of the chest has been exposed, the interscapular region and the lateral chest walls should be similarly mapped out and exposed. After a preliminary series of 30 such exposures a few days should be allowed to elapse, and a second shorter series of 4 or 5 exposures should be begun. This should be repeated three or four times with a period of rest between the period of exposures. Clopatt uses a similar method, but makes the interval of rest as long as ten days. There is no doubt that the effect of the X-ray treatment would be increased by means of a compression apparatus such as that devised by Jessen and Rzewuski, which diminishes the susceptibility of the skin and allows of the use of a more powerful X-ray.

As stated above, the type of growth which yields most satisfactorily to X-ray treatment is the so-called lymphosarcoma. However, Pfahler has observed a decided improvement in a number of cases of secondary mediastinal carcinoma following mammary cancer. In one case of this kind reported by him the patient survived six years after the treatment.

The results in these cases are due to the destruction of the tumor cells with definite diminution in the size of the growth, as demonstrated by the X-ray shadow and the physical signs, and the consequent disappearance of the pressure symptoms. In some patients a decided improvement in the symptoms has been noticed after the second session, and in most of those who reacted well the dyspnea and cyanosis disappeared to such an extent that the patient was enabled to return to work. As far as can be judged from the literature, a permanent cure has never resulted. All of these patients eventually relapse. In the case of lymphosarcoma amelioration may last as long as two years. When relapse occurs a second regression may occasionally be produced by X-ray treatment, but, as a rule, all curative treatment is ineffectual. In some cases radiation produces absolutely no effect. Radium may produce even more satisfactory results than X-ray according to Burnham and others. Considerable amounts are necessary, 1 gm. or more. All but the Gamma-rays should be filtered out and a fixed distance from the skin should be established.

Operative interference, in the form of resection of a portion of the sternum or of portions of one or two ribs over a tumor presenting externally, seems justified in some cases, from a palliative point of view. Such a procedure is comparable to decompression in an inoperable brain tumor. It may relieve the symptoms due to compression of the structures within the mediastinum, render the patient decidedly more comfortable so far as dyspnea is concerned, and prolong life.

The relief of dyspnea, which is the most distressing symptom in mediastinal growths, is always indicated. The possibility of relieving it depends upon its cause. If due to general pressure, resection of the ribs or sternum may be indicated as described above; if partly due to pleural effusion, as is often the case, removal of the fluid by tapping must be considered. Powell suggests that complete removal of the fluid is inadvisable, as the altered pressure conditions so produced actually favor increased rapidity of growth in the neoplasm, while, on the other hand, the positive pressure of the fluid retards growth. For this reason it seems advisable to remove just enough fluid to relieve the urgent dyspnea. When the difficulty in breathing is due to the inclusion of the trachea or bronchi in a mass of neoplasm and the subsequent direct pressure, operative interference is of little avail. Tracheotomy has been occasionally performed, and it has sometimes been feasible to introduce a long tube through the tracheotomy wound beyond the point of compression, but there is grave danger in such cases of ulceration of the growth, with infection spreading to surrounding tissues, or producing perforation. In most cases a resort to drugs is necessary. Inhalations of oxygen produce relief in some patients. No rules as to dosage can be laid down; some patients take very large quantities with apparent benefit. Powell recommends a mixture of iodid of sodium (gr. iii—0.2 gm.) and chloral (gr. v to vii—0.3 to 0.5 gm.) every three, four, or six hours. When paroxysms of dyspnea occur, Abram advises the inhalation of chloroform, but this must certainly be used with great caution, as the patient is so dependent on the accessory muscles of respiration that if these are paralyzed a fatal result may ensue.

For the relief of severe pain in mediastinal tumor, as in other conditions, opium in some form is the only drug of real value. When the pain is not very severe, the deodorized tincture orally administered may suffice and is desirable on account of its less constipating effect. If the pain is severe, morphia hypodermically will be needed.

The sleeplessness from which many of these patients suffer may, in the early stages, be relieved by veronal, trional, or sulphonal, but later, especially if associated with dyspnea and pain, chloral or a mixture of morphia and chloral will be necessary.

MEDIASTINITIS

Suppurative Mediastinitis.—Inasmuch as suppurative mediastinitis sometimes results from operations in the lower part of the neck, great care should be exercised in such operations to avoid infection. When foreign bodies gain access to the esophagus or trachea these may, if neglected, cause ulceration and mediastinal suppuration, and for this reason they should be removed as promptly as possible.

During the early stages of mediastinitis, before actual suppuration has occurred, the application of ice to the front of the chest by means of the ice-bag, or applications of heat by means of compresses or poultices, should be made in the hope that the inflammation will subside without suppuration taking place.

The treatment of suppurative mediastinitis, after pus has formed, is based upon the principles which govern the treatment of empyema. If it is suspected that the disease has suppurated, but no demonstrable changes on the surface of the chest are apparent, an exploratory aspiration with a large needle done under local anesthesia is justifiable.

In a certain number of cases the treatment is comparatively simple, for the pus points between the ribs. In such cases the treatment is exactly the same as for empyema. An incision is made over the fluctuating area, the pus is evacuated, and, to insure a permanent opening for free drainage, a piece of rib 5 or 6 cm. long is resected and a drainage tube is introduced. If it has been determined by aspiration that pus is present, but the exact situation and extent of the pus formation are doubtful, trephining of the sternum must be resorted to. This is a very old procedure, and was successfully carried out by Galen. After a trephine opening has been made, exploration can be carried on through this. If it is found that the trephine opening is above the most dependent portion of the pus pocket, a counteropening must be made either by trephining the sternum a second time or by resecting a portion of rib so that thorough drainage may be established. If, as is apt to occur with tuberculosis, abscesses, etc., the sternum itself is diseased, the affected portion of the bone must be removed. Milton has reported a case in which almost the entire sternum was removed in this way with subsequent recovery of the patient.

In some instances, where the superior portion of the mediastinum is affected, the abscess points in the neck, either above the manubrium or one of the clavicles. In these cases, simple incision and evacuation often suffice to relieve the urgent symptoms, but seldom allow free enough drainage. A counteropening should also be made in these cases, although

Lerche states that by using suction the necessity for a counteropening may be avoided in some cases. In connection with Pott's disease or penetrating foreign bodies from the esophagus, it may be necessary to open the posterior mediastinum. When this occurs in the superior portion it may be possible, as Haidenhain has shown, to reach the pus pocket from the neck. When the inferior portion of the posterior mediastinum is involved, a more difficult procedure is required. Faure recommends the resection of the first rib posteriorly near its spinal attachment. This, he claims, allows a wide opening into the posterior mediastinum. Other surgeons recommend resection of the transverse processes of several vertebræ. The great danger in such an operation appears to be death from suffocation due to an extrapleural pneumothorax, but this should be avoidable if the operation is done under differential pressure.

TUBERCULOSIS OF THE MEDIASTINAL LYMPH NODES

Prophylaxis.—The prophylaxis of tuberculosis of the mediastinal lymph-nodes is that of tuberculosis in general, though it must be remembered that the patients will be mostly children.

While it has not been proved that tuberculosis of the mediastinal glands is commonly due to bacilli of the bovine type, pure milk should be insisted upon. The common source of infection is no doubt droplet and dust infection from careless adults, so that the education of infected adults is of great moment in prophylaxis.

In the house, owing to their low stature and their almost constantly dirty hands, children are especially likely to be infected from contaminated carpets or floors. The carpet should be banished from the sleeping room, and, when feasible, from the other rooms largely occupied by children, and moist sweeping and dusting should be insisted upon. The children's sleeping room should contain plenty of fresh air, and should be properly screened against flies, as, indeed, should the whole house.

Children who are under weight, anemic, or suffering from local disease which predisposes to tuberculosis infection should receive special attention. Adenoids and enlarged tonsils should be removed. The underweight and anemic children should be placed in open-air schools and should be encouraged, as should healthy children, to indulge in outdoor games. Such encouragement is needed especially by girls. The diet should be simple, but nutritious and easily digestible. Eggs, milk, butter, cream, cereals, the easily digestible vegetables, and meat in moderation should form the basis. When possible, children should have an annual change of scene to a climate somewhat different from that to which they are usually accustomed.

Treatment.—A radical cure of tuberculous lymph-nodes in the mediastinum by surgical methods has seldom been attempted, and, on account of the frequency of spontaneous recovery, and the difficulty of accurate diagnosis, even with the X-ray, scarcely seems justifiable, as a rule. Nevertheless, there are instances where the enlarged glands threaten life from pressure on vital structures, when surgery is justified. Occasionally it has been possible, even when life was not endangered, to remove tuberculous bronchial glands with comparative ease. Such a case has been reported by Thiemann. The cases in which surgical interference is warranted will, however, be few in number.

The medical treatment may be general and supportive or symptomatic. The general supportive treatment relates mainly to the hygiene of living, and differs at different stages of the disease.

The climate which has been found best suited to patients with glandular tuberculosis is the seaside, and, when change of climate is desirable, and possible, this should be chosen. The patient should be in the fresh air twenty-four hours a day, and, if not confined to bed, may be allowed, if old enough, sea-bathing in moderation provided the baths are brief, are followed by a brisk rub with a rough towel, and result in good skin reactions. In recent years valuable results have been obtained by heliotherapy.

Children under two years of age, and very thin, delicate children, of greater age, do not bear sea-bathing well; for them baths of heated salt water are of benefit. The temperature of the bath for such children should not be below body heat. In inland climates baths of artificial sea water, or salt rubs, are of value.

Rest.—A proper degree of rest is essential, even when there is no fever or other signs of marked toxemia. When the maximum temperature regularly goes above 99.5° F. or 100° F., or there are definite signs of toxemia, absolute rest is demanded. In cases where the patient is able to be about there should be a definite period of rest in the middle of the day, and plenty of time for sleeping at night.

Diet.—The food should be easily digestible, rather rich in fats, and abundant in quantity. Care must be taken, however, not to stuff patients to such an extent that the digestion is upset and the metabolic processes are overtaxed in burning up unnecessary waste. The patient should be systematically weighed at least every two weeks, as this is one of the best indices of progress.

Drugs.—Of the drugs which have been empirically proved of value, iodids, especially in the form of syrup of the iodid of iron, stand first. Arsenic is of value in some cases. The hypophosphites and phosphates are highly recommended by some. Cod-liver oil is still highly recommended; it is now known that it contains a large amount of fat soluble vitamin and it is this that distinguishes it from other oils of equal caloric value.

Forchheimer obtained good results from creosote (benzosol) before meals with syrup of the iodid of iron after meals.

Tuberculin.—If the diagnosis is perfectly clear, specific therapy by means of tuberculin should be tried, if the daily maximum temperature is not above 99.5° F. or 100° F. It is especially advisable in mediastinal gland tuberculosis, because ordinarily surgical interference is seldom justified. The dose to be used must be very carefully determined on account of the well-known great difference in susceptibility to tuberculin. As little as 0.00001 mg. may be sufficient to cause a reaction, and, although reactions occur less easily than in pulmonary tuberculosis, violent ones are certainly to be avoided. Whether Wright's method of employing very small doses, insufficient to cause a reaction, and increasing them but little, or the German method of beginning with small doses and increasing them considerably is most desirable is an open question. Good results have been obtained by both methods. The frequency with which tuberculin should be given is also a question open to discussion—certainly not more than twice a week. The value of the opsonic index, unless carried out by one constantly doing the work and skilled in the technic, is dubious. The practitioner with ordinary resources had better depend on the vital signs and the general condition of the patient. The variety of tuberculin to be used is a matter of opinion. Many consider the old tuberculin as valuable as its successors. Some use bacillin-emulsion, which is less toxic. L. Brown advises a mixture of equal parts of bacillin-emulsion (B. E.) and broth filtrate (B. F.). In the administration of tuberculin it should be diluted with 0.25 per cent carbolic acid in normal saline. The dilutions deteriorate after a couple of weeks. In order to insure accuracy a regular tuberculin syringe should be used, the needle should be small and sharp, and the injection should be introduced beneath the skin of the interscapular region. No skin preparation except cleansing with alcohol or the application of iodin is necessary.

Symptomatic Treatment.—Symptomatic treatment may be necessary in some patients. Fever should be combated by bathing. A salt sponge (5 per cent salt) at 90° F. may be given at night, and can with advantage be followed by an alcohol rub. If the temperature is not reduced a cold pack to the trunk is advisable.

Some authors advise the reduction of fever by drugs, such as aspirin or novaspirin, lactophenin, or pyramidon. All of these are unsuitable for continuous use, and, if lactophenin or pyramidon are given, their effect on the heart and blood should be remembered.

Dyspnea, if urgent, must be treated as in other mediastinal tumors. Tracheotomy with passage of a tube beyond the obstruction is justifiable as a temporary procedure at times. Inhalations of oxygen and the use of the various sedatives mentioned under mediastinal tumors may be tried. Dysphagia may be temporarily relieved by the passage of sounds, but this is a questionable procedure, as rupture of a softened gland may occur. It is in cases with urgent dyspnea or severe dysphagia that radical operations should be considered.

INDURATIVE MEDIASTINOPERICARDITIS

Up to a few years ago the treatment of mediastinopericarditis was the treatment of valvular heart diseases, for the effect of binding the heart to the anterior chest wall and the surrounding structures is, in the long run, to cause failure of compensation, demanding the usual treatment for this condition (see Chronic Myocardial Insufficiency, p. 227).

Surgical Treatment.—The introduction by Brauer in 1902 of the operation, termed by him "cardiolysis," opened a new field in the treatment of mediastinopericarditis. Reasoning from the point of view that it was the rigid chest wall which was mainly responsible for the extra work thrown upon the heart muscle, Brauer suggested resection of a portion of the chest wall overlying the heart, with the idea that the greater freedom of action thus obtained would lead to improvement in the cardiac musculature. This hope has, in the limited number of cases so far operated upon, been realized to a considerable extent.

According to Brauer and others who have had success with the operation, the procedure should only be attempted if the heart still shows evidence of a good degree of competency. If there are a well-marked systolic retraction, a diastolic shock, and general indications that the break in compensation has not reached an advanced stage, the operation is permissible.

The name "cardiolysis" gives altogether a false impression of the gravity of the operation, even to the professional mind, inasmuch as it indicates actual manipulation of the heart itself. As a matter of fact, the operation is not much more complicated than the ordinary operation for empyema, and, of more than 20 cases now reported, only 1 has resulted in death directly referable to the operation. The term "precordial thoracolysis" is preferable, we think, to cardiolysis, or perhaps "precordial thoracostomy" would be even better.

Technic.—The technic of the operation now varies slightly from that originally proposed by Brauer, but is essentially unchanged. The operation can be easily done under local anesthesia in cases in which a general anesthetic is contra-indicated. A semilunar flap with its convexity toward the median line is made to the left of the sternum in such a position that the costal cartilages and the adjoining parts of the third, fourth, and fifth ribs may be exposed. The flap should, of course, include all the soft parts down to the bone, and should permit the exposure of at least 10 or 12 cm. of the cartilages and ribs mentioned. It is now considered sufficient to remove subperiosteally from 6 to 10 cm. of each of the third, fourth, and fifth costal cartilages and adjoining ribs. Bleeding is then controlled and the flap is replaced. The procedure is, therefore, a very simple one as now advised, and differs from some operations previously done in that no attempt is made to remove any portion of the sternum, and removal of the perichondrium and periosteum is not considered necessary. While technically new bone might result with the periosteum left behind, the experience gained from König's case shows that the amount of bone formed has no deleterious effect; on the other hand, removal of the periosteum adds greatly to the difficulty of the operation, increases the likelihood of shock, and adds the danger of injury to the heart and perforation of the pleural cavity. Inasmuch as the operation has been performed in a number of cases in which indurative mediastinopericarditis was not present, the suggestion of William MacKenzie, that a portion of the fifth rib be removed first to form an exploratory opening, is worth considering. As this author points out, if we are unable to feel the intrapleural area through such an opening we know that adhesions exist.

Results of Treatment.—The results of the operation described above are, as a whole, very satisfactory. In some cases the operation has been undertaken at a period when the damage to the heart muscle was too great to be even temporarily repaired; in such cases failure has naturally resulted. Of 8 cases collected by Roux-Berger in 1910, 5 resulted fatally. Of these 1 was not mediastinopericarditis and 2 were also suffering from endocarditis with valve leaks. The ultimate results in many patients cannot be followed up in the literature, but in many the immediate results were very striking; often the heart became more regular as soon as the ribs were removed, and the subjective symptoms diminished markedly a few hours after the operation. Recession of the signs of cardiac incompetency in remote organs took place more gradually, but was often progressive, and in many instances the patient was able to return to work after a protracted invalidism previous to the operation. Several of the patients remained in good condition one year after the operation, and one survived as long as four years, and died of miliary tuberculosis. The operation is, of course, of no value if the adhesions are to the spinal column or the diaphragm.

REFERENCES

Dermoid Cysts

Eiselsberg, von. Arch. f. klin. Chir., lxxi, 575, 1903.
Morris. Med. News, lxxxvii, 404, 1905.
Nordmann. Deutsche med. Wchnschr., xxxii, 205, 1906.

SURGERY OF MEDIASTINUM

Amburger. Beitr. z. klin. Chir., xxx, 770, 1901.
Faure. Johns Hopkins Hosp. Bull., xvi, 125, 1905.
Stoyanov. Rev. de Chir., xix, 388, 1899.

SUPPURATIVE DISEASE

Kopfstein. Wien. klin. Rundschau, xvi, 861, 1902.
Milton. Lancet, i, 872, 1897.

X-RAY TREATMENT

Clopatt. Deutsche med. Wchnschr., xxxi, 1150, 1905.
Elischer, von, and Engel. Deutsche med. Wchnschr., xxxii. 1620, 1906; Ibid., xxxiii, 505, 1907.
Grob. Cor.-Bl. f. schweiz. Aerzte, xxxix, 232, 1909.
Herringham. Clin. Journ., xxxv, 33, 1909-1910.
Kienboeck. Wien. med. Presse, xlvi, 2377, 1905.
Pfahler. Penn. Med. Journ., ix, 649, 1905-1906.
Schwarz. Wien. klin. Wchnschr., xx, 1468, 1907.
Zum Busch. Tr. Clin. Soc., xxxix, 232, 1906.

INDURATIVE MEDIASTINOPERICARDITIS

Lecene. Arch. d. mal. du cœur, ii, 673, 1909.
Roux-Berger. Semaine méd., xxx, 423, 1910.
Thorburn. Brit. Med. Journ., i, 10, 1910.
Venus. Centralb. f. d. Grenzgeb. d. Med. u. Chir., xi, 537, 1908.

DISEASES OF
THE CIRCULATORY SYSTEM

DISEASES OF THE HEART

CHAPTER VIII

DISEASES OF THE PERICARDIUM

ALEXANDER McPHEDRAN AND WILLIAM FLETCHER McPHEDRAN

History.—Before taking up the treatment as generally practiced at the present time it will be of interest and of no little advantage to look briefly at what was done for pericarditis after it was first discovered to be a distinct disease.

Its earliest treatment is well presented in the writings of Latham, New Sydenham Society (1789 to 1874), in the early part of last century, and, later, by those of Sir Thomas Watson.

Bloodletting, mercury, and opium were the remedies advised in all diseases of the heart, in acute rheumatic fever, and in fact, in all other acute infectious illnesses. "In the treatment of acute rheumatism, it might be laid down as an axiom to treat the rheumatism and let the joints take care of themselves. But where there is a question of the heart being affected it is the latter which must receive attention. Mercury to salivation is indispensable to the cure of every case of pericarditis" (Latham).

His pupil and successor, Sir Thomas Watson, regarded the disease as one of great importance because of the danger of speedy death from the great quantity of effusion which often occurs. Following the teaching of his time, he regarded bleeding as the most potent means of arresting the disease. This was the view of the most distinguished both at home and on the Continent. Bouillard declared that by the bold use of the lancet it is possible to extinguish the inflammation and jugulate or slaughter the disease at its birth. Watson had found that this could only result if the copious bleeding were done before the to-and-fro friction sound occurs, but not after. But he thought that, even after the to-and-fro friction sound is heard and adhesions are beginning to form, repeated free cupping of the precordium may do good. At the same time the patient was to be brought under the influence of mercury as soon as possible, that the gums might swell and become tender. It was found, unfortunately, in this as in other diseases that the system frequently resisted with great obstinacy the influence of mercury. He therefore urged that calomel should be given in frequently repeated doses, guarded, if need be, by opium from the first. When the gums began to rise there were always a prompt subsidence of distress and mitigation of the symptoms—less pain, less palpitation, and less dyspnea.

In cases with high temperature and dry skin, tartar emetic, gr. 1/6 to 1/4 (0.01 to 0.15 gm.), was given, to cause free perspiration. If the pulse was strong and full a large blister was applied to the precordium.

It was soon recognized that this heroic treatment should be reserved for the plethoric and vigorous patients, and that the debilitated should be treated much less vigorously. This led in time to the recognition of the fact that "many seem to recover under any treatment, or even with none at all" (Watson). It is remarkable how keenly alive at this early period these master minds were to the danger of future adhesions in all cases of pericarditis. They warned against being too sanguine as to permanency of recovery, as months or even years may elapse before secondary effects are evident. "No medium between complete cure and sudden death" (Latham). How confident they were of the efficacy of their heroic treatment is shown by Watson's faith in it. "I am of opinion that by the cautious employment of the lancet and leeches, and by early and unshrinking use of mercury, the mischief may be greatly limited and the consecutive changes staved off to a distant period." And yet he recognized that many cases of pericarditis recovered "under any treatment or with none."

PERICARDITIS

Prophylaxis.—As acute rheumatic fever is much the most common cause of pericarditis, it is evident that when once an attack of this infectious illness of unknown etiology has occurred in a child, the utmost care should be exercised to prevent a recurrence. Acute rheumatic fever is an acute infectious disease of unknown etiology, exhibiting arthritis, angina and affection of the heart. Whilst the means of obtaining information as to the endocardium and pericardium are available to every physician employing the ordinary methods of physical examination, namely, percussion and auscultation, it is for some unknown reason true that he is ever apt to ignore the vastly more important myocardium. The importance of bearing in mind, at all times, the heart muscle is a matter upon which too much insistence cannot be laid.

Children who have undergone an attack of acute rheumatic fever, are not only likely to have recurrences, but these recurrences are likely to have associated with them grave evidences of cardiac disability. The danger of cardiac infections decreases rapidly as the child grows older, and at all times the endocardium is more apt to be the seat of the disease than the pericardium. Occasionally one finds pericarditis the only, as it may be the first, manifestation of an attack of rheumatism in a child. But when rheumatism or chorea has occurred once, even if the attack is very slight, the physician must use every effort to prevent recurrence of the attack by a careful examination of the gateway of infection—the tonsil. It is not enough to decide that because the tonsils are not so hypertrophied as to meet in the center line they are not at fault. Care should be taken to have the examination of this portal of infection thorough. The glands of the neck are often an excellent guide, for in the case of chronic infection of the tonsils drained by them, they are to be found enlarged, perhaps tender, and becoming more normal as one approaches the clavicle. Dysphagia in an infection of the upper respiratory system is the most important factor in the history. As the individual swallows, the anterior and posterior pillars are approximated, and so pain is experienced in the inflamed tonsil. On examination, it is often found that there are abnormal vessels running over the surface, and that on pressure over the anterior pillar, a quantity of liquid pus is evacuated from the crypts or from the sinus between the anterior pillar and the tonsil.

Much mischief often comes from small, deep-set tonsils, whose crypts are plugged with mucus and degenerating epithelial cells. These are very apt to become infected or to be subacute infected areas at all times. The tonsils should be examined by probing the crypts while pressure is at the same time applied outside on the neck. Any tonsil that is the seat of chronic inflammatory change should be removed, not amputated by a guillotine, but dissected out. Too much attention cannot be devoted to this almost universal point of infection in the rheumatic child. Similarly, all local areas of infection from which possible pericarditis might arise are to be treated carefully. The need for such precautions will, of course, be greater if former attacks of pericarditis have occurred. It is doubtful if an attack of pericarditis can actually be prevented, but diseases of which pericarditis is a part or complication are well within the bounds of preventive medicine.

In the mildest attack of acute rheumatic fever, even when there is only a slight "growing pain," the child should be put to bed. Not only the rest, but the warmth to be had in bed, may prevent, or, at least, moderate, the severity of the attack. At the same time, the heart beats less rapidly, and so the liability of extension of the rheumatic process to the cardiac structures is reduced materially. The rheumatic fever should be energetically treated so that as early a recovery as possible may be expected. Once the heart has been affected its resistance to a new infection is reduced, and so new attacks occur the more readily.

The prophylaxis of pericarditis, then, depends in acute rheumatic fever, as in other diseases, on a preservation of the bodily health. Should any disease occur, by keeping the patient quiet both bodily and mentally, there will be less danger of pericardial involvement.

Treatment.—In all cases of acute rheumatic fever and other infectious illnesses, the physician's attention will be drawn more particularly to the heart, and so to the pericardium, by increased pulse rate while at rest, with a further disturbance upon movement and dypsnea. These it must be remembered are the symptoms of myocardial insufficiency. When there is pericarditis present, one would expect to find precordial distress, and possibly a to-and-fro friction rub. Then absolute rest in bed should be enjoined. Probably the hope of mitigating, if not preventing, an attack is vain in most cases presenting such premonitory symptoms. The patient should be supported comfortably on pillows. He will probably be more comfortable semirecumbent on the left side, but his comfort and lack of dyspnea must be the best indications of the excellence of this position. The bed should be firm and narrow, so that the patient may be easily moved by the nurse or attendant. The use of a double inclined plane, such as is used in surgical beds for the maintenance of the Fowler position, will be of very considerable comfort and benefit to the patient. The back may be raised at will to any height desired. Such a bed as described by Gatch is in the writers' experience excellent. He should not be permitted to stir for food, voiding of urine, or movement of the bowels. This position in bed should be maintained until the acute symptoms have subsided and until the circulation is efficient, and the heart not unduly accelerated on getting up for short increasing periods of time. Sir William Gull once went to see a patient with an old house officer. The man had a pericarditis, which the physician had not recognized. He was expressing his chagrin at the oversight to his old chief when the latter

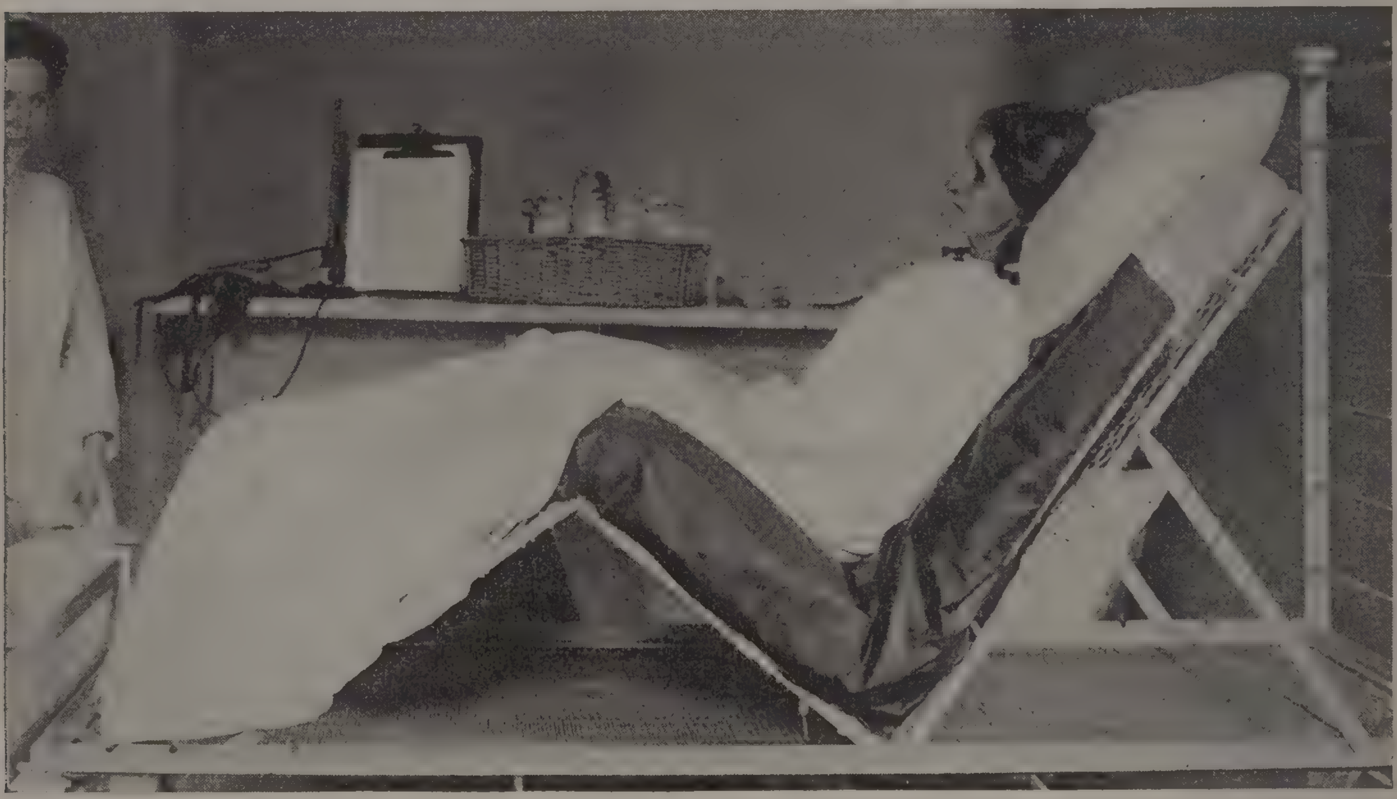


FIG. 1.—THE GATCH BED.

patted him on the shoulder and said: "Never mind, my lad; had you recognized it, you might have treated it."

This sums up the treatment of the mild cases of pericarditis.

In cases in which some or all of the physical signs of pericarditis are found on careful repeated examinations, it has been said that moderate movements by the patient should be permitted, as they aid in preventing adhesions by causing the heart to alter its position with each new posture. This is a doubtful inference, and in any case the benefits from it must be too slight for it to be wise to run the risk of other dangers for the doubtful good to be gained. In cases in which the weakness, disturbed action of the heart, and increase in the area of precordial dulness make it probable that there is considerable myocarditis and some effusion, complete rest is of the utmost importance, as sudden movements, especially changing to the erect posture, may cause faintness or even a fatal collapse. Here the symptoms are due to the myocarditis. It has been suggested that the action of the heart may be inhibited by the inflammation of its serous covering just as is the case with the intestine in peritonitis. This is very doubtful, for in fatal cases the cardiac muscle is always found to be profoundly affected. So constantly is this the case that it is doubtful if even large effusions can do more than hamper the action of a moderately diseased heart muscle, but the hampering might be sufficient to turn the scale if the myocardium were severely, although not fatally, damaged. If there is cough it should be subdued by repeated doses of codein, gr. ¼ (0.0515 gm.), and heroin, gr. 1/12 (0.005 gm.). There should be no straining at stool. To secure a soft and easy motion a plain enema may suffice; if not, some mild laxative, such as sulphur, olive oil; or, if a more active drug is needed, compound licorice powder. Castor oil answers well, and is more effective, but should not be given if the stomach is irritable, as the excitement of the heart's action by vomiting should be avoided. It is highly probable in view of the fact that the patient is taking a light diet without much residue available for colonic irritation, and so for natural movements, that less disturbance will be caused by an enema. Here there is only one disturbance while with the most carefully judged cathartics there may be several.

Diet in Acute Pericarditis.—In mild cases the patient may have whatever he desires, with no restriction, except as to the amount of fluid. In the more severe cases it is of greater importance that the food should be attractively prepared, and always given in small quantities. This method of feeding prevents overloading of the stomach with the distressing cardiac symptoms arising therefrom. In addition, one must remember that the stomach is liable to rebel against the quantities of salicylates which the medical attendant will use in the treatment of the common rheumatic form of this disease. The diet should be simply and easily cooked (for great demands are to be made upon the household) and soft, so as to be easily digested as well as to avoid irritation to the inflamed tonsils. There should not be too much residue for bowel evacuation. While soft, the nourishment must be fairly concentrated, since highly diluted food means the introduction of a large amount of water. This tends to increase the work of the heart and also provides fluid for a possible or existing effusion. For this reason a wholly milk diet is inadvisable. Soft-cooked eggs, soft toast, scraped beef done into balls with a little butter and broiled are valuable additions. Kephir and koumiss are of particular use, because they are easily digested, and on account of the lactic acid bacilli (of which they are cultures) are good antidotes to meteorism. It will be advantageous to increase the nutritive value of the milk by the addition of cream, and of the milk and kephir by the addition of lactose—say, ʒi (gm. 4) with each feeding. It is tasteless, high in nutritive value, and shows slight tendency to fermentative changes. The milk can be given as cocoa or in the form of soups, flavored, if desired, with vegetables, or it might be advantageous to give gruels of well-cooked oatmeal, with cream, to replace one of the milk feedings. The eggs are best cooked by placing them in a warm bowl, covering them with boiling water, and letting them stand covered for five minutes. A small amount of seasoning is permissible, but, while the whole problem of exudation and edema in relation to salt intake is under investigation, much salt should not be allowed. The number of calories in this diet is quite sufficient to maintain the body for some time in bed, and as the patient progresses the diet may be increased. But for some little time the administration of frequent small meals—say five per day—will save the heart a great deal of unnecessary work, because there will be easier digestion and less danger of distention of the stomach and intestines.

The following table would be satisfactory for some time. The milk items might well be given as milk soups, because there is the danger of the patient rebelling against a monotonous dietary. The fluids total 1,400 c.c., and there is ample room here for about 200 or 300 c.c., that is, ℥vi, of water, which had best be given between feedings or in sips of cracked ice. To allay the thirst a trace of lemon juice might be added, but due regard must be paid the alkaline treatment secured through the copious doses of the bicarbonate of soda. To give water more freely would tend to dilate the stomach and increase the probability of effusion.

Diet—

7 A. M.—Milk. 200 c.c. ℥vii.
Cream. 50 c.c. ℥ii.
Lactose. 4 gm. ʒi.
9 A. M.—Soft eggs. 2.
Toast. 2 pieces, 10 to 15 gm. each.
11 A. M.—Kephir. 200 c.c. ℥vii.
Lactose. 4 gm. ʒi.

1 P. M.—Scraped beef. 50 gm. ℥ii.
Toast. 2 pieces.
Custard made of: milk, 200 c.c., ℥vii; eggs, 2; sugar, 4 gm., ʒi.
3 P. M.—Kephir. 200 c.c. ℥vii.
Lactose. 4 gm. ʒi.
5 P. M.—Soft eggs. 2.
Soft toast. 2 pieces.
Milk. 100 c.c. ℥iiiss.
Lactose. 4 gm. ʒi.
7 P. M.—Kephir. 200 c.c. ℥vii.
Lactose. 4 gm. ʒi.
9 P. M.—Milk. 200 c.c. ℥vii.
Cream. 50 c.c. ℥ii.
Lactose. 4 gm. ʒi.

				Calories
Totals:	Milk + Kephir,	1,300 c.c.	=	884
	Cream	100 c.c.	=	250
	Lactose	28 gm.	=	106
	Scraped beef	50 gm.	=	100
	Eggs	6	=	450
	Bread	12 gm.	=	40
				1,830

Local Treatment.—The application of *cold* to the surface over the heart is, as a rule, the most effective local treatment. In recent years the local use of ice in deep-seated inflammations has become more and more general. When it first came into use it aroused much prejudice. It was regarded as unpleasant and likely not only to increase pain, but also to cause extension of the inflammation. Experience has proved that these fears are groundless and that, on the contrary, the pain is usually relieved, and the inflammation lessened or even arrested. Ice produces both of these results with much more certainty than hot poultices, and is much cleaner. The ice-bag offers, as a rule, the most convenient means of applying cold. It may be applied directly to the surface of the precordium. If the direct application is too severe the bag may be wrapped in a flannel cover or have a piece of flannel placed under the ice-bag. In this manner the cold can be tempered, and as the surface becomes accustomed to it the flannel may be removed. In cases in which it is inadvisable to apply extreme cold, as in children and old people with feeble circulation, Leiter's tubing may be substituted and the water used gradually lowered in temperature to any desired degree. In all cases, especially where there is precordial distress or oppression, it will be well to have the bedclothes raised on a cradle. The ice-bag or Leiter's tubing may be suspended from this so as to take all possible weight off the sensitive region.

We do not know how cold produces the effect. Probably it acts by causing contraction of the arterioles of the parts in relation to the surface to which it is applied. It may stimulate the vasoconstrictor nerve or possibly the muscular walls of the vessels themselves. For example, dipping the hand in cold water causes actual diminution in size of the radial artery. That is, the area to be supplied with blood can control, by its sensory stimuli, the amount of blood brought to the area. Sir Lauder Brunton has shown that ice applied over the sternum of rabbits causes blanching of the bronchial mucous membrane, and that, if the cold is continued, the vessels dilate and the membrane becomes hyperemic. Remission of the cold application causes a new vasoconstriction and blanching. Until the mode of action is proved by investigation, we must rest content with theories.

Local bloodletting may be resorted to with benefit, especially in the robust. It is best done by applying eight to ten or more leeches over the cardiac area, and after their removal the bleeding may be encouraged by warm fomentations. This procedure usually relieves pain, and often probably lessens the tension in the chambers of the heart when they are overdistended. The abstraction of blood must have some direct influence on the distention of the vessels and heart. But the relief cannot be wholly due to that; for, if so, the application of a bandage around the thigh tight enough to obstruct the venous flow, while allowing the arterial flow to enter, should produce greater effect. It seems reasonable to infer that the irritation of the leeching should cause reflex contraction of the distended vessels of the pericardium. This would cause more rapid flow of blood, that is, a better circulation, the pain being relieved by the lessening of pressure of the distended vessels on the nerve terminals and the increased force of blood flow aiding the heart to expel its contents into the pulmonary vessels.

Blisters to the precordium is an old method of treatment for the relief of pain and distress, but it is little resorted to now. It is more severe and not more successful than are the means above described. Some, however, prefer the application of a series of small blisters which are allowed to heal up rapidly. Their action must be due to the effect of the irritation of the surface on the vessels of the pericardium. Their use in effusion, which is slow in absorption, may be wise, because in some cases their application is rapidly followed by removal of the fluid.[1]

Drugs.—Of drugs we have none that are specific in pericarditis. Bleeding, mercury, and opium, the sheet-anchors of the practice of our forefathers, having failed to cure, or even probably to affect favorably the course of the disease, have long fallen into disuse. As the acute form of pericarditis is but one of the local manifestations of a general disease, we should endeavor to control the local through treatment of the disease on which it depends. The majority of cases are due to rheumatic infections; of the remainder most are due to grave forms of septic infections as pneumonia and septicemia. Drugs are of little if any avail.

---

[1] The galvanocautery or Paquelin cautery has been recommended. If counter-irritation is indicated at all, it can be applied in this way with an amount of precision impossible in any other way.—Editor.

For the treatment of acute rheumatic fever we have in the salicylates a drug which is, if not a specific, at least one that has marked influence on its development and course. It is necessary to give it sufficiently freely to bring the system under its full influence as soon as possible, that is, until it produces slight toxic effects as shown by *tinnitus aurium.*

I am strongly of the opinion that the quantity usually given is not adequate for the purpose in view—the arrest of the disease if possible, and, if not, the mitigation of its progress. For this purpose about 20 gr. (gm. 1.3) of sodium salicylate given every two hours will be required to act promptly; or even 40 to 60 gr. (gm. 2.6 to 4) as a first dose and followed by the smaller quantity.

Unfortunately, salicylates frequently irritate the stomach and cause vomiting; this effect is probably due in most instances to the breaking up of the salt. Vomiting, even nausea, does grave injury to a patient whose heart requires the greatest possible rest. To prevent the setting free of salicylic acid, 30 to 40 gr. (2.0 to 2.6 gm.) of bicarbonate of soda should be added to each dose of the salicylate, or potassium citrate may be used instead; bicarbonate is a useful addition in the treatment of acute rheumatic fever. The earlier such treatment is begun in the rheumatic attack the more likely is it to prevent infection of any of the structures of the heart. After the heart has become infected the administration of the salicylates is regarded by many able observers as, in the first place, being without value because they exercise no influence on the cardiac infection, and, in the second place, dangerous from their depressing effect on its action, since in every case the myocardium is infected and therefore liable to dangerous depression. It is doubtless true that the salicylates fail to influence the progress of some cases of pericarditis, just as they unfortunately fail in not a few cases of polyarthritis. Possibly this failure is because the infection in these cases is not rheumatic and so not amenable to salicylate treatment. Then the more frequent failures in cardiac infections may be due to the partial withholding of the salicylates on account of the fear of dangerous depression of the heart, a fear that I have not in my experience seen to rest on good grounds. Only pure salicylates should be given. The natural is usually best tolerated and least likely to cause vomiting. Salicin, much used in England instead of the salicylate, is found less effective, probably because less soluble.

Sodium cacodylate, 0.18 gm. (3 gr.), hypodermically administered, has been used by many for acute rheumatic fever for many years. It is said to be advantageous particularly when the disease is severe enough to cause such reactions as pericarditis. I have not been impressed favorably with the effects and feel that the results so obtained have not been so good as those with salicylate treatment.

As in acute rheumatic fever so here the treatment should be continued for some days after both symptoms and signs of pericarditis have been lessened or disappeared. It is better to continue the salicylates for too long than for too short a time.

Other treatment is not commonly required in pericarditis. The ice-bag usually relieves the pain and distress, and aids in reducing the fever, which, however, is rarely high. If the ice-bag, for any reason, is not well borne, it may be necessary to replace it with hot applications. These sometimes give the patient greater relief than cold, just as they do occasionally in pleurisy. Unfortunately they excite the action of the heart and their use should therefore be avoided if possible. If the pain continues, small doses of opium, as in Dover's powder, gr. 1 to 5 (gm. 0.06 to 0.3), according to the symptoms usually suffice to give relief and, at the same time, quiet the irritability of the patient. If the action of the heart is irregular and tumultuous and the fever high, quinin in 3 to 5 gr. (0.2 to 0.3 gm.) doses, repeated three or four times daily, usually does much good. In nervous patients a bromid may be needed and usually brings the needed quiet. Veronal, trional, and sulphonal have no effect upon the pain, hence do not give good sleep. Chloral is dangerous because of the unpleasant cardiac symptoms occasionally following its use.

Stimulants.—Persistent disturbed action and weakness of the heart are doubtless due in all cases to the associated myocarditis and require cardiac stimulants in addition to the greatest repose. Of the cardiac stimulants digitalis is the most effective; it should be given early, as soon as the first symptoms of disturbance of the heart's functions appear. The object of the digitalis treatment is to depress the rate at which the heart beats. This effect is best obtained when fibrillation of the auricles exists. Then the circus movement of the auricular musculature is broken as Lewis has shown, and there is a possibility that regular rhythm will be restored. This is followed in its turn by lengthened periods of diastole—the phase of the heart muscle activity when it rests, renews its nourishment and allows the blood and lymphatic circulation to remove the waste products. In cases in which there have been chronic affections which weaken the heart, as valvular lesions, chronic nephritis, chronic anemia, etc., digitalis should be given very early, before the heart shows any symptoms of disturbance. Of course, due regard must be given to its effect on the preexisting chronic affection. If chronic degeneration has already occurred in the wall of the heart from the old disease, the additional infection incident to pericarditis may cause marked irregularity of the heart's action and other symptoms of failure of its power. In such cases often neither digitalis nor other stimulant steadies or relieves the symptoms. Here morphin, gr. 1/8 to 1/4 (0.008 to 0.015 gm.), given hypodermically at bedtime, is usually followed by a good night's rest and much steadier action of the heart. Should this dose of morphin be ineffectual it should be repeated every half hour till the patient secures the desired effect. Morphin by inducing mental and physical rest is the best of all heart stimulants. The myocardium so rested has better tone, contracts better and so carries out its work. Then digitalis will have full effect in restoring the efficiency of the heart as far as that is possible.

In acute cardiac failure with collapse, dyspnea, and much precordial distress associated with pericarditis, strophanthin given intravenously may give prompt relief. In a man of fifty-five, with much distress the pulse being about 140 and very irregular in rhythm, 1 mg. caused the pulse to fall to 75. It became almost regular, and was followed by complete relief of the urgent symptoms. The transition was almost alarming to the physicians; a smaller quantity might have been ample for relief, although less striking in its effect.

After Treatment.—Convalescence should be slow. The patient can be raised to a sitting posture for half an hour each day, and the time gradually increased. Should there be no ill results he can stand upright in a week and walk a few steps. During this period careful observation of the pulse rate, with daily inspection of the apex beat and cardiac dulness, will determine his progress. In the prevention of adhesions a good course of cardiac exercises with the observance of due care to prevent strain is essential. Various courses of gymnastics have been recommended and are discussed under the head of Diseases of the Myocardium. The principal feature to be kept in mind is that they all should be supervised by a physician. Passive movements and, above all, massage are of particular benefit. Such exercises should be carried to the stage of gentle fatigue only. Carbonic acid baths have also been recommended—with what success is at present uncertain. In the cases where there has not been myocarditis of an extensive degree, it is quite within the bounds of possibility to expect a complete return to normal.

Pericarditis with Effusion

If moderate and the heart shows no signs of embarrassment, no special treatment is required, since, as a rule, the effusion soon becomes absorbed. The patient should be placed in bed with complete rest, so as to relieve the heart of work as far as possible. If the effusion increases it soon fills the pericardial cavity so that the walls of the heart become subjected to positive pressure. This would soon lead to compression of the flaccid auricles, and, to a slight extent, lessen the dilating force of the ventricles, and therefore their suction power. Such a condition makes the heart's work inefficient, and is therefore characterized by a degree of dyspnea, precordial distress, and cyanosis in proportion to the derangement of function. Since the heart is unable to dilate properly, especially the auricles, to receive the blood, little, if any, benefit can be obtained from the administration of digitalis or other cardiac stimulants. The diuretics which act directly on the kidneys, such as caffein, diuretin, and theocin-sodium acetate, may increase the urinary outflow greatly and prove of much service. At the same time, the amount of liquid taken should be reduced to as low a quantity as the patient is able to tolerate.

Owing to the debilitated condition of the heart in all cases of pericardial effusion, due to the myocarditis as well as the hampering of its action by the pressure of the exudate, no treatment which in any way depresses its function should be resorted to; hence diaphoresis, by hot baths of any kind, by hot packs, and by pilocarpin, is a dangerous depressant, and not permissible. For the same reason we should not attempt to reduce the effusion by free purgation.

Paracentesis.—If, after due trial of these measures, the effusion persists, resort should be had to paracentesis. If the quantity of fluid is only moderate and not increasing perceptibly, there need be no haste to remove it, but, if large and increasing, its early withdrawal is necessary, especially if the heart becomes weak, irregular, and its contractions increase in frequency. Under such circumstances the pressure of the fluid is not only obstructing the circulation by disturbing the action of the heart, but also by interfering with the coronary blood supply. The diminished coronary supply favors the increase of the degenerative changes in the structures of the heart, present in greater or less degree in all cases of pericarditis. In estimating the danger from the presence of the fluid, we have to consider three aspects of the matter: first, the quantity of the fluid; secondly, the rapidity of its exudation; and, thirdly, the degree to which it has embarrassed the function of the heart, and of these the third gives the most important signals of danger; since, if the heart itself is much damaged in its muscular structures, the pressure of a very small quantity of fluid may be enough to disturb its function dangerously.

In cases of moderate effusion and moderate disturbance of the heart action, we may content ourselves with withdrawing an aspirating syringeful of fluid for diagnostic purposes. Such a procedure may lead to the absorption of the residue similar to the process observed in pleuritic effusions. There being no urgent symptoms we may wait a few days before taking further steps. Then, if there is no improvement, the fluid should be removed by an aspirator, or a trocar and cannula, to which is attached a long tube to act as a syphon. The needle of the aspirator should have a sheath into which it can be withdrawn as soon as the cavity is entered in order to prevent the heart from being wounded. If the symptoms have been urgent, the relief following the removal of the fluid is very striking and gratifying. Paracentesis of the pericardium was first proposed by Riolan in 1649, and afterward by Senac in 1794.

The operation was very slow in being recognized as justifiable, not to say advisable; as late as 1870, Billroth characterized it as a prostitution of surgical skill. Since then it has come to be recognized as the only efficient means of treatment in persistent effusion, and as imperative in all cases in which the exudate at all disturbs the function of the heart.

The operation causes only moderate pain, and therefore the danger of general anesthesia should in no case be incurred. Local anesthetics give moderate relief and serve the purpose fairly well. Of these the infiltration method with dilute novocain solution is preferable. But freezing the area with ethyl chlorid or even the degree of cold obtained with ether or ice and salt will serve the purpose in most cases.

Technic.—Various sites are recommended as the seat of puncture, the difference of opinion arising chiefly from the varying views as to the position of the heart in the distended sac. Being of greater specific gravity than the fluid, it was formerly taken for granted that the heart sank to the most dependent part, that is, to the back in the recumbent or even the semirecumbent position, the fluid filling the anterior part behind the precordial surface. Later observations have shown that this is often not the case; that in many cases of large effusion the heart lies in contact with the anterior chest wall, or nearly so. In a lad of twelve, from whose pericardial cavity I withdrew over 1,000 c.c. (35 ounces) of effusion due to tuberculous disease, the heart was touched by the sheathed needle almost as soon as it had entered the cavity. Many similar cases have been reported. There is no single point of choice which is preferable or even suitable for all cases. The several points recommended, beginning to the right, are:

1. The right fifth intercostal space, in the cardiohepatic angle into which the pericardium forms a triangular projection, and which early becomes filled in pericardial effusion. Rotch has been especially insistent on the advisability of selecting this site. Puncture has also been advised in the third and fourth right intercostal spaces close to the sternum.

2. The angle formed by the ensiform cartilage and the left costal margin. This is the site preferred by many of the leading British and American writers. It was suggested by Larrey in the early part of the last century, but not regarded seriously (Dock).

If the needle is entered close to the costal margin and, as soon as the point reaches the posterior surface of the cartilage, is turned sharply upward and to the left behind and in contact with the posterior surface of the cartilage, it will enter at once the cavity of the pericardium, especially if the latter is depressed by effusion. If carefully done there is no danger of puncturing the peritoneum, liver, or pleura. It further affords an excellent guide to subsequent incision and drainage of the cavity if such should be advisable. As this is the point I have always selected, I am able to speak for both its safety and efficiency. It should be at least no more liable to accident than is any other available point; some risk attaches to each of them.

3 and 4. The fourth or fifth left intercostal space, either close to the sternum or 2.5 cm. (about 1 inch) further to the left. In the former point there is much danger of wounding the internal mammary artery. In the second, the heart, if it lies near the anterior wall of the chest, is liable to be punctured, and that, too, in its thinnest and least contractile part, the right auricle. This would be a suitable and efficient site if there was assurance that the heart lay toward the posterior part of the cavity.

5. The fifth or sixth intercostal space outside the left mammary line at a point midway between the cardiac impulse (if it can be made out) and the outer limit of dulness; if the cardiac impulse cannot be determined, then a point about an inch internal to the left limit of dulness may be chosen. This is advised as a safe point so far as injury to the heart is concerned; the fluid, if present, is easily reached. Even if the heart is struck, it is in its thickest part, the left ventricle, which can scarcely be penetrated completely unless adherent to the wall of the chest. The pleura may be punctured, but with a sterile needle such an accident will cause no damage. If pleural effusion exists, it can also be removed as the cannula is being withdrawn.

The choice of the site is not a vital matter; they have all given good results and the choice will therefore depend on the special conditions to be dealt with, such as the shape of the precordial dulness, the position of the heart if it can be determined, etc. It is wise, first, to do an exploratory puncture. The patient should be in the recumbent position or propped up comfortably with pillows. Having determined that fluid can be reached, an aspirating needle or trocar and cannula may be used; it should be firmly but slowly pressed in until the change in resistance is perceived, when the pericardial sac is entered. This is usually possible, unless there is much dense plastic exudate lying in contact with the sac. The size of the needle or trocar and cannula required will depend on the density of the exudate. For serous exudate one with 1-mm. bore will be sufficient. For all thicker exudates the bore must be larger. Cole has suggested that in tapping a pericardium where there is reason to believe that the effusion is in the posterior part a fine rubber catheter should be passed inside the cannula. When the cannula has entered the sac the catheter is pushed forward and its softness enables it to follow the pericardial curves freely and without damage to the structures. Thus the back of the sac may be reached easily.

Failure to obtain fluid by puncture at any of these sites is not an infrequent experience. This may be due to various causes, such as absence of free fluid, the exudate being wholly fibrinous; a thick layer of fibrinous deposit on the wall of the sac; plugging of the needle by the fibrinous exudate, or a mass of fibrin floating in the fluid; the loculation of the fluid at the posterior part of the sac, etc. A "dry tap" may be, and probably often is, due to the absence of fluid, the increased area of precordial dulness, even when very great, being due to a greatly enlarged heart covered by a thick layer of plastic exudate. That this is true is shown by the case of a young man who entered the Toronto General Hospital with marked signs of pericarditis. The precordial dulness was very large, extending nearly to the midaxilla, and there was dulness with blowing breathing at the back below the left scapula. Two inches (5 cm.) inside the left border of dulness faint cardiac impulse could be felt. There was also dulness in the right cardiohepatic angle. The symptoms of cardiac embarrassment were very marked—dyspnea, a weak, very rapid pulse, livid facies, perspirations, and faintness on any movement being made. It seemed certain that the pericardial sac was distended with a copious effusion. An attempt at aspiration was made in all the points described in the following succession—the left xiphocostal angle, the right fifth intercostal space, the left fifth intercostal space close to the sternum and an inch to the left, and halfway between the weak cardiac impulse and the left margin of dulness. No fluid was obtained at any of them, and the needle on penetrating 2 or 3 cm. within the pericardial cavity came into contact with the heart in all of them, except the one to the right of the sternum. There can be no doubt that the greatly distended pericardium was due to an enormously dilated heart covered by a thick layer of plastic exudate. Large doses of digitalis and of camphor were given, and within a day improvement began and progressed rapidly. He made a complete recovery. He returned two months later for examination; he showed no abnormal signs in connection with the heart and was well and strong. Time alone will prove whether serious adhesion of the pericardial surfaces will occur. However, the prompt improvement which followed the administration of full doses of digitalis renders it probable that the infection did not seriously involve the areolar tissues around the pericardial sac.

Hydropericardium

This condition occurs in the course of chronic nephritis, tuberculosis, or other cachectic conditions. It may result from some local obstructive pressure, such as tumor of the pericardium, or it may be due to an occlusion of the mouths of the lymph vessels by a subacute inflammation of the endothelial cells. It frequently occurs after scarlet fever. The academic question as to whether the effusion is a transudate, or due to a local inflammatory reaction, is, fortunately, without practical interest, because the treatment is not influenced by such a minute differentiation.

It is probable that unless the effusion is so great as to cause urgent dyspnea and tachycardia it will not be discovered, unless careful physical examination is carried out daily. The main efforts in treatment must be directed to the principal disease, to which the pericardial effusion is secondary. It is possible that something might be expected from such good diuretics as digipuratum (Knoll) or theocin-sodium acetate, or the two combined. Cathartics cannot be recommended in the depressed state of the cardiac muscle. But if the condition has progressed to such an extent as to be clearly recognizable, paracentesis alone will probably be efficacious. Solution of adrenalin (1:1,000), 1 c.c., may be injected after paracentesis in the hope of arresting the exudate. In the patient's weakened condition, and with the knowledge that it will almost certainly have to be repeated, the greatest care to maintain asepsis will be necessary.

In the light of recent treatment of old tuberculosis resistant to sanitarium treatment by artificial pneumothorax, it is interesting to note that Wenckebach has lately advocated removal of the pericardial effusion, and subsequently the introduction of sterilized air into the sac. He reports the case of a man twenty years of age, who had developed tuberculosis of the left apex, and who shortly after admission showed marked evidences of dyspnea, tachycardia, cyanosis, with signs of effusion into the pericardium. Thirteen hundred c.c. were removed, and after seventeen days 1,500 c.c. more. Following this second aspiration he had a severe hemorrhage from the left lung. His condition necessitated another aspiration, but the fluid returned so quickly that little could be hoped for from this procedure. Wenckebach had been in the habit of following all thoracentesis with the introduction of air, claiming that by so doing greater amounts of exudate can be removed; that there are not such acute changes in intrathoracic pressure, and that thoracentesis can be freely performed on cases where it would be otherwise impossible. At the next aspiration air was admitted to the pericardium by the apparatus used for artificial pneumothorax. There was not so rapid a return of the effusion, it lost its hemorrhagic characters, and, after two more aspirations with the introduction of air, the patient was so much improved that he was able to leave the hospital. He came back to the clinic, but when another aspiration was done and no air admitted he soon complained of rapid filling of the pericardium with its attendant distress. Seven months after the ninth aspiration, when only 300 c.c. were removed, he was found to be much improved; no effusion was demonstrable, he breathed well, and, while his liver was much enlarged, he had good thoracic movements on respiration. The actual existing phthisis seemed improved.

It is only natural for one to consider that such a maneuver had saved his life, for previously the exudate quickly recurred and aspiration was followed by speedy pulmonary hemorrhage.

This is only an isolated case, but, when the large number of patients afflicted with pulmonary phthisis have had new life and hope offered to them by the analogous operation of artificial pneumothorax, it is only right to offer a sufferer from tuberculous pericarditis a chance of having such an operation. He has nothing to lose and everything to gain.

Pyopericardium

In pericarditis occurring in the course of pneumonia and septic infection and in some cases of tuberculosis the exudate is usually purulent from its onset. Pus in the sac is an abscess, and must be treated as such surgically. An incision and free drainage are necessary. The incision is made about 3 cm. to the left of the sternum, or, probably better, along the lower margin of the left seventh costal cartilage in the epigastrium and upward through the attachment of the diaphragm into the cavity of the pericardium, as advised by Cyril Ogle. This incision, being free and at the lowest part of the pericardium, permits of free drainage and of easy exploration and removal of fibrinous masses. It is well to remember that localized encapsulated collections of pus are as commonly met with in the pericardium as in the pleura. But the need for this grade of incision is not as striking as is the case when the pleura is involved, for here the interspaces are wider proportionately and there is less movement on respiration.

A paracentesis alone is not justifiable. The pus must be cleaned out thoroughly. In cases due to a local infection alone much could be hoped for from the injection of 1 or 2 c.c. of a 2 per cent formalin solution in glycerin. This has been followed by excellent results in several cases of purulent pleurisy observed by me. When the cases are part and parcel of a general septicemia the outlook is naturally very unfavorable. A vaccine prepared from the exudate may prove useful after drainage.

Hemopericardium

Most cases of blood in the sac are due to serious internal hemorrhage. This may be from rupture of the ascending portion of the aorta, of the heart or of the coronary arteries. Hence the flow of blood is so rapid that all the usual means of treating internal hemorrhage are of no use, the patient dying before they can be applied.

The recent improvements of surgical technic in thoracic surgery offer a more hopeful outlook when the condition is traumatic in origin. If the injury is severe and the flow of blood great, the patient will die of cerebral anemia or of pressure of the extravasated blood upon the heart.

Cases of hemorrhagic exudate are to be treated as cases of hydropericardium if sterile and if the exudate does not clot. Otherwise they must be dealt with as pyopericardium.

Pneumopericardium

As it is exceedingly rare to find pneumopericardium unassociated with pus in the sac, the treatment must be the same as that of pyopericardium. Some authors advise the removal of the air by aspiration. This would relieve the heart temporarily. The condition and prognosis depend upon the primary disease. As it rarely arises, save late in the course of some severe constitutional disease, like carcinoma of the esophagus with rupture into the sac, or a rupture of a pyopneumothorax, the prognosis is grave.

Adhesive Pericarditis

Acute pericarditis, whether dry or with effusion, may heal completely, leaving as little trace as does an ordinary pleurisy. If the process is long and drawn out, the probabilities are that there will be vascularization and growth of granulation tissue through the exudate. This process may result in adhesions between the visceral and parietal layers of the pericardium and between the pericardium and other organs. It is the extrapericardial adhesions which cause injury to the heart. Several cases, observed clinically and at autopsy, have shown that no damage results from obliteration of the cavity, if there are no important adhesions to the surrounding viscera or the chest wall. These extrapericardial adhesions may then be (1) to the diaphragm, (2) to the pleura, (3) to the posterior mediastinum, and (4) to the ribs, cartilages, and sternum. In the treatment of the first three the adhesions cannot be removed, and as the structures are all soft there should not be intense opposition to cardiac work as in the case of the fourth class. In these cases accordingly the prophylactic treatment of cardiac failure is indicated, that is, work physical and mental, only to the stage of general slight fatigue; good hours; gentle systematic exercises and avoidance of all strain; digitalis and strychnin if followed by any improvement. Probably anemia will develop early in the course of the disease. This can best be treated with iron and regulation of diet. Cases of polyserositis can only be treated by aspiration of the effusion as it occurs in chest or abdomen.

In the treatment of the fourth class of extrapericardial adhesions, Brauer, of Marburg, had Peterson do an operation to relieve the condition. Brauer proposed "to relieve the heart functionally by breaking the strong, bony ring of ribs, not by a severe operation with the breaking up of extensive adhesions, but only by substituting a soft covering for the natural bony covering of the heart. The operation was tried upon a patient with adherent pericardium, broken compensation, ascites, and edema. Segments of the third, fourth, and fifth ribs, 7 to 9 cm. in length, were resected under light narcosis, the periosteum being carefully removed. His pulse became stronger and more regular, the ascites and edema disappeared, and he was able to do heavy work without symptoms. The pulse still remained irregular." Brauer showed 2 cases at the meeting of the International Medical Congress at Budapest in 1909. In one the result was excellent; in the other only moderate relief resulted because the heart muscle was much injured by the inflammation. To date I have been able to find 32 cases; 24 of these have shown marked improvement and in some instances this was evident, even during the course of the operation.

The myocardium must be in good condition. As the fibrosis resulting in adhesions takes place there is a similar process infiltrating the musculature of the heart, which, it will be recalled, is always involved to some degree in pericardial inflammation. The physician must satisfy himself that this has not proceeded to an extraordinary degree. To ascertain the actual condition of the heart muscle, the patient should be observed in bed, and under the effects of exercise and drugs. Persistence of unmodified dyspnea, cyanosis, and edema are contra-indications to the performance of the operation. I feel that in cases where a failure of circulation has occurred, and there are present evidences of well-marked adhesions to the chest wall, that is, tugging in of the lower ribs, fixation of the left border of cardiac flatness on inspiration, immobility of the cardiac apex on change of posture, fixation of precordial region on inspiration, the operation should be brought to the attention of the patient and relatives at an early date. Surely, with these physical signs, especially if corroborated by a good radiologist, we need not wait till the failure that is bound to come occurs. If the operation is performed in a quiescent period, the chances of improvement are much greater than if during an exacerbation of the chronic heart failure. This quiescent heart period, moreover, makes the choice of an anesthetist much easier. The greatest attention should be paid to the selection and administration of the anesthetic to make the shock as little as possible. Schlayer is of the opinion that several cases of polyserositis have been subjected to this operation without adequate care being taken in the differential diagnosis. These cases naturally are not suitable ones for Brauer's operative interference.

The operation itself is fairly simple. A horseshoe-shaped incision is made over the third rib, extending from the sternum to the mammillary line, and downward to the sixth or seventh rib. The pectoralis, and all structures above it, are turned upward in a flap. Then the third, fourth, fifth, and sixth ribs and cartilages, or as many as will be found implicated, are removed for four or five inches from the sternum. The periosteum behind the ribs is left to avoid injuring the pleura. Some surgeons prefer removing the periosteum by dissection after the rib excision; others destroy the osteogenetic properties by cauterization. Some have pointed out that, as most of the cases are young and the cartilages are still uncalcified, there will be no growth from perichondral tissues. This matter must be left to the discretion of the surgeon doing the operation.

As postoperative care, a long convalescence with a course in cardiac gymnastics is much to be desired.

REFERENCE

Ogle, Cyril. Lancet, i, 693, 1900.

CHAPTER IX

ACUTE AND SUBACUTE ENDOCARDITIS

EMANUEL LIBMAN

SUBACUTE BACTERIAL ENDOCARDITIS

Subacute bacterial endocarditis generally attacks valves which are the seat of previous disease. The lesion upon which the disease is engrafted is usually rheumatic, much less often syphilitic, and still less often atherosclerotic. A congenital malformation such as a patent ductus arteriosus, or the wall of the aorta or the pulmonary artery, may be the site of localization of the infection. In many instances the patients were unaware of a previous cardiac lesion. It is of interest to note that patients with valvular defects, showing marked symptoms of decompensation, or suffering from fibrillation, rarely develop the disease.

SYMPTOMS AND PATHOLOGY

As a rule, the onset is insidious. The patient may go about for months without realizing that he is seriously ill. The initial symptoms are extremely varied, the most common being lassitude, weakness, anorexia, headache, dizziness, cough, pains in the bones and joints, chills, or chilly sensations and fever. The symptoms of myocardial insufficiency are rarely the earliest. Occasionally the disease is ushered in by the manifestation of some embolic accident.

The symptomatology of the disease can best be appreciated from a knowledge of the pathological findings. The vegetations are of variable size, irregular in shape, and friable in consistency. Their color is greenish, pinkish-gray, red, or yellowish. When situated on the mitral valve, they rarely have a tendency to block the orifice. They are not limited to the line of closure as in the cases of endocarditis due to rheumatic fever, but involve the valve above and below it, generally affecting the free edge. The vegetations characteristically spread upward along the posterolateral wall of the left auricle and downward along the chordæ tendineæ, at times causing ulceration and rupture of the latter. When the aortic valve is involved, the vegetative masses may be very small or quite large and have a tendency to spread downward on the mural endocardium in the region of the septum membranaceum and along the ventricular aspect of the aortic cusp of the mitral valve. Aneurysms of the mitral and aortic valves and of the septum membranaceum may be produced. There is but little tendency to ulceration, and rupture from one cavity of the heart to another has thus far not been seen. Involvement of the tricuspid valve is exceedingly uncommon, and the pulmonic valve has in the absence of an open ductus arteriosus never, in the author's experience, been affected. The vegetations in what is known as the active or bacterial stage contain myriads of bacteria which are responsible for the disease. The anhemolytic streptococcus (the so-called Streptococcus viridans) is found in 95 per cent of the cases, most of the remaining ones being due to the Bacillus influenzæ (Pfeiffer's bacillus). The clinical picture in the cases due to the influenza bacillus is the same as that found in the streptococcus cases, and what we said of the latter holds true for the former.

An analysis of the symptoms of subacute bacterial endocarditis shows that they can be classified into two groups. Firstly, there are the symptoms due to the valvular defect as such or as influenced by the anemia and exhaustion or the complications of the disease. These may be spoken of as the myocardial symptoms. Secondly, there are the symptoms which are due to the infection. These are of two kinds, those due to the toxemia, and those due to the vegetations on the valvular and mural endocardium. The toxemia is responsible for the fever, and its attendant phenomena, including the anemia. The liberation of small or large fragments of the vegetative masses into the blood-stream results in a group of symptoms which we have termed the "endocarditic symptoms." The latter include embolisms to various organs and the extremities, the tender cutaneous nodules (Osler nodes), the embolic aneurysms, the petechiæ, and purpura.

Myocardial Symptoms.—The myocardial symptoms are the same as those occurring during the course of any valvular or myocardial disease. These include such symptoms as dyspnea, palpitation, cyanosis, cough, hemoptysis, thoracic pain, visceral congestion and edema of the lower extremities. As a rule, they are not marked during the active stage of the disease. In many cases there are no evidences of myocardial insufficiency until late in the disease. Murmurs are generally present; in rare cases, they may be absent for a long time and, when this occurs, it is due either to the small size of the vegetations, their distance from the free edge, or their presence exclusively on the wall of the left auricle. It is unusual for new murmurs to develop during the course of the disease. Auricular fibrillation seldom occurs. Fibrinous pericarditis is not part of the disease.

Fever.—Fever is the most constant symptom. At first it is generally low and, in the milder types of the disease, it may remain low. In a great majority of the cases, as the disease progresses, the fever gradually seeks a higher level and becomes irregularly remittent or at times intermittent. From time to time, there may be brief periods when the fever is either low or absent, although the bacteria are still in the blood. A sharp rise of temperature may follow embolism. Chills and chilly sensations are common. At times, the association of fever and chills leads to an erroneous diagnosis of malaria. Sweats are present in nearly all the cases. Lassitude, anorexia, and loss of weight are practically always present.

Anemia.—Anemia is a constant symptom, and is generally progressive. The hemoglobin is often less than 65 per cent, and may fall to 20 per cent. The anemia is always of the secondary type, unless nitrogen retention be present. The skin of the face often gradually assumes a *café-au-lait* color, which when fully developed is quite characteristic.

Emboli.—The embolic phenomena give rise to the most distinctive symptoms of the disease. They include the emboli to the various organs and the extremities, the petechiæ, the hemorrhages in the fundi, the Osler nodes, the embolic aneurysms, and the focal embolic lesions in the kidney. It is not within the scope of this article to discuss in detail the clinical findings resulting from embolism to the various intra-abdominal and thoracic organs, the brain and the extremities.

Petechiæ.—Petechiæ are found in over 80 per cent of the cases. Those with white centers are the most characteristic. They are particularly to be found in the skin of the lateral aspects of the neck, in the supraclavicular fossæ, and in the conjunctival and buccal mucous membranes. They also occur in the fundus of the eye.

Osler's Nodes.—The tender erythematous nodes of Osler are believed to be almost pathognomonic. They are found not only in the terminal phalanges of the fingers and toes, and the thenar and hypothenar eminences, but also between the webs of the toes. At times they are represented by firm anemic areas, which are best found by palpation. The tenderness of these nodes is their most important characteristic.

Aneurysm.—Embolic aneurysms occur here more frequently than in any type of endocarditis. Any artery may be affected, those most commonly involved being the arteries of the brain and of the extremities. When an embolic aneurysm of one of the cerebral arteries ruptures, subarachnoid or intraventricular hemorrhage may result, giving rise to a syndrome simulating meningitis. The so-called serous meningitis or polymorphonuclear meningitis, with or without the streptococcus being present in the spinal fluid, may occur in the course of this remarkable disease.

Pain, Tenderness and Osseous Lesions.—Tenderness of the lower sternum is a symptom of value when present. As will be seen later, it is more prominent in the bacteria-free stage than in the active stage. Pains in the joints are common and slight swelling may occur, but the joints are never greatly swollen and red as in rheumatic fever. Clubbing of the fingers is very common, and should be looked for in every case. It is, by itself, not diagnostic.

Spleen.—Enlargement of the spleen is an important symptom. Infarctions are very frequent and may give rise to pain in the splenic area, the epigastrium, the precordial area, or in the left shoulder. Nausea and vomiting may be present, and, rarely, hematemesis. Here, as in gross embolisms to other organs, there may be a sharp rise in temperature and often a chill. The liver may be enlarged and tender.

Kidney.—The urine often contains albumin and casts. A characteristic finding is the presence of microscopic hematuria. The latter is due to embolic focal glomerular lesions in the kidneys. In the presence of infarction due to plugging of a branch of one of the renal arteries, marked albuminuria results and often macroscopic hematuria. In a few instances, acute glomerular nephritis has occurred. As will be seen later, chronic glomerular nephritis is an important finding in the bacteria-free stage.

Blood.—Blood cultures are of the greatest importance in the diagnosis. They are positive in over 90 per cent of the cases. For details of the morphological, cultural and biological characteristics of the anhemolytic streptococci which were grown from the blood in 95 per cent of our cases, and the methods of obtaining cultures, the reader is referred to a paper by Libman and Celler (*Am. Journ. Med. Sc.,* 1910). Repeated blood cultures should be taken. In cases with chills and repeated high elevations of temperature, it may not be possible to grow the bacteria despite their presence in great numbers in the vegetations. Following intravenous medication of any sort, the patient may experience a chill and the temperature may drop to normal for a few days. Hand-in-hand with such a reaction, the blood cultures may become negative for several days.

DIAGNOSIS

Bacterial Stage.—It may be useful to enumerate the findings which are of greatest diagnostic value in the active or bacterial stage of the disease. They are the presence of an old lesion of one or more valves, prolonged fever, progressive anemia, embolisms to the various organs and extremities, white-centered petechiæ, hemorrhages in the fundi, the Osler nodes, embolic aneurysms, splenomegaly, tenderness of the sternum, the *café-au-lait* color of the face, clubbing of the fingers, microscopic hematuria, and the positive blood cultures.

Bacteria-free Stage.—Most of the patients with positive blood cultures die with bacteria still present in the blood, the usual duration of the disease being from four to eighteen months. Some of the patients with positive blood cultures, while under observation, lose the bacteria from their blood, but later die from the sequelæ of the disease. This constitutes the bacteria-free stage. At autopsy, these cases show vegetations in the characteristic locations, but they have undergone fibrosis, hyalinization and calcification, and living bacteria are no longer found in them. In a large number of these cases, the patient is already in the bacteria-free stage when first seen by the physician. It should be emphasized that negative blood cultures alone do not establish the diagnosis of the bacteria-free stage. There are individuals showing the typical symptoms of the active stage in whom the blood cultures remain persistently negative, and yet at the post-mortem examination, the vegetations are found to contain enormous numbers of living bacteria. Such cases are merely instances of subacute bacterial endocarditis in the active stage with negative blood cultures.

The bacteria-free stage is characterized not only by the failure to obtain a positive blood culture, but by the absence of living bacteria from both the vegetations and the blood stream, and by healing of the vegetations. Clinically, the patient is not cured; he generally succumbs to the damage done to the heart valves, myocardium, blood-forming organs, and kidneys during the period when the bacteria were present in the blood.

Although the vegetations no longer feed bacteria to the blood, they are still capable of giving rise to embolic phenomena. The latter are a great deal less marked than in the active stage. On the other hand, the myocardial symptoms are often more severe. Petechiæ occur, but are neither as abundantly nor as frequently present as in the bacterial stage. The Osler nodes occur infrequently, and embolic aneurysms are likewise very uncommon.

Fever may be present, but is generally not as high as in the active stage. Following embolism, there may be a sharp rise of temperature, although the vegetations no longer contain bacteria. Fever may also occur due to anemia or intercurrent conditions. Many of the patients, on the other hand, have practically no fever during the bacteria-free period of their illness. An appreciable degree of anemia is generally present. The hemoglobin value is usually less than 65 per cent, and in the most severe cases may fall below 20 per cent. As a rule, the anemia is progressive. The leukocytes are usually not increased, and leukopenia is not infrequent. In many cases the percentage of polymorphonuclear leukocytes falls as the disease progresses.

The spleen usually remains enlarged and palpable. At times it reaches such great proportions as to cause confusion with a primary splenic disorder. The association of marked splenomegaly with leukopenia has in some instances led to a mistaken diagnosis of Banti's disease.

Tenderness of the sternum is generally more marked in the bacteria-free cases than in the active ones. Pains in the bones and joints are not as frequent and the sweats and loss of weight not as marked as in the active stage.

The renal phenomena in the bacteria-free stage are of special interest. Albuminuria and cylindruria, as in the active stage, may be present. Likewise microscopic hematuria may occur, but neither as frequently nor as persistently as in the bacterial stage. A certain percentage of the bacteria-free cases develops symptoms of renal insufficiency due to the presence of chronic diffuse nephritis. The renal symptoms may be so marked as to dominate the clinical picture in a certain number of the healed cases. Death often occurs in uremia. Other causes of death in the bacteria-free stage are anemia and exhaustion, myocardial insufficiency, cerebral embolism, and intercurrent infection.

In certain cases, positive blood cultures become negative and, in addition to the disappearance of the bacteria from the blood, there is a complete recovery. In such cases, the only symptoms which remain are those due to the original valve defect. It is not known how often complete recovery occurs, but it is probably a great deal more frequent than we are at the present in a position to estimate. When I had completed the observation of the first 150 cases of the disease, I had already observed 4 cases of complete recovery. Since that time I have seen at least 2 more such cases. There is unquestionably a large number of mild cases which recover without having been observed while the disease was active. The cases reported by Oille, Graham, and Detweiler, are interesting in this connection. These patients had slight fever and murmurs; some had petechiæ and a large spleen, and in all the so-called Streptococcus viridans was isolated from the blood. At the time their paper was written, all seemed to have recovered, but a sufficient length of time had not elapsed to determine the final outcome of these cases. It is important to note that recurrences of the disease have been observed.

From time to time various authors have reported cases of recovery from even relatively severe forms of the disease and have attributed the favorable outcome to some particular therapeutic procedure. In considering the results of a given form of therapy, especially if tried in a small series, it must be borne in mind that spontaneous recovery has been known to occur in severe cases.

TREATMENT

Vaccines and Serums.—Various types of vaccines and serums have been used in the treatment of the disease. These include stock streptococcus vaccines, non-specific vaccines, autogenous vaccines, dried extracts of vaccines, sensitized vaccines, anti-streptococcus serum, serum of human beings and animals previously immunized against the organisms isolated from the patient's blood, and convalescent serum. Galloway states that he saw encouraging results following the use of serum obtained from a donkey previously immunized against the patient's own organism. The further history of the patient is not reported. A few cases of recovery have been reported in which vaccines were held accountable for the favorable outcome. Latham and Hunt reported a case in which recovery was attributed to the use of autogenous vaccine administered by mouth. Blood cultures had been positive six times. Hemsted reported a case with recovery in which a variety of vaccines and serums were employed. Before the blood cultures became positive the patient was given Marmorek's antituberculous serum, polyvalent streptococcus serum, and streptococcus vaccine prepared from the expectorated blood. No effect was noted. Later, when the anhemolytic streptococcus was given from the blood, autogenous vaccines were given by injection in conjunction with the oral administration of a special serum prepared by immunizing a horse against a mixed culture of streptococci and the patient's own streptococcus. The patient finally recovered. Horder tried vaccine therapy in 6 cases due to anhemolytic streptococci and in 2 due to the influenza bacilli. In 2 of the streptococcus cases, there was some reduction of the fever and improvement of the general condition, but neither patient recovered. One of the influenza bacillus cases improved following the use of autogenous vaccine and was originally reported by Horder as cured. Later, the patient relapsed in spite of a third course of vaccine treatment. Jochmann treated 2 of his cases by the combined use of autogenous vaccine and antistreptococcus serum. One of the patients recovered. A third patient recovered without the use of either vaccine or serum. The subsequent histories of these cases are not known. Billings, who reports a case of recovery, considers vaccines and serums of no value. Debré employed vaccines and immunized horse serum without observing any beneficial influence. Fette had no success with autogenous vaccine. Four of our 6 cases of recovery were treated by autogenous vaccine, in 3 injected subcutaneously and in 1, intravenously. So many cases have been treated with vaccines and serums by other observers and ourselves without result, that one is not warranted in ascribing the favorable outcome in any of the reported cases of recovery to the use of these agents.

Chemical Antidotes.—A large number of drugs has been used in an effort to cure the disease. Among them are the colloidal metals, especially those of silver, for example, collargol, argochrom, dispargen, mercury compounds, arsenic derivatives, phenol, formalin, the sulphocarbolates, quinin, and salicylates. These drugs have been administered orally, subcutaneously, and intravenously. An effort was made by Epstein to treat the disease by the intravenous injection of colloidal iodin. Although the drug was found to have no curative value, the blood was sometimes freed from bacteria for as long as four days following sharp reactions. Recent additions to our therapeutic armamentarium are neutral acriflavin, mercurochrome, gentian violet, and rivanol. Our experience up to the present time with the first three of these remedies, in a small group of cases, has been entirely unfavorable. Further studies will be made. With mercurochrome one must be ready for sharp febrile reactions and marked gastric disturbances.[1] In weak patients, it should not be employed in therapeutic attempts. It is important to watch the leukocyte count when gentian violet is administered. It may cause sharp depressions, amounting at times to leukopenia. Of all the drugs mentioned, the arsenical preparations seem to be the most worthy of trial. Lorey ascribes recovery of one patient to the intravenous use of arsphenamin and arsacetin. One of our cases of recovery (a streptococcus infection of a luetic aortic valve) was treated with arsphenamin. A number of other cases, however, have been treated with arsphenamin, without favorable result.

The studies of Capps with sodium cacodylate are well worth repeating. He reports the use of this drug in 8 cases with positive blood cultures, and of these, 6 are still living. One patient is well after eleven years; 2 patients, after five and five and a half years respectively; one, after two years and three months, and 2 patients were improving after five and six months of treatment, respectively.

The patients who were treated successfully by Capps in this way were apparently suffering from a mild form of the disease, but none the less, the large percentage of recoveries makes the method of therapy worthy of trial. Capps advises the daily intravenous injection of sodium cacodylate in doses of from 1 to 5 gr., the average dose being 3 gr. The drug should be pushed until a strong garlic odor is exhaled from the breath. The solution should be freshly prepared every week or ten days from a reliable preparation of the drug, the commercial ampules being unsatisfactory for the purpose. Injections should be continued daily for a period of months if necessary. He advises absolute rest in bed during the entire period of the treatment. Ill effects were not noted. If diarrhea develops, the drug should be discontinued for several days. Whether this method will be found to be of value in the severer cases remains to be ascertained. As Capps points out, other arsenical preparations may prove to be of greater efficacy than sodium cacodylate.

Transfusions.—Transfusions are of value not so much for any specific bactericidal effect of the injected blood, as for the general improvement which results in the patient's condition. In patients in whom severe anemia is present, many of the symptoms which the patient experiences are aggravated by the presence of the anemia. Following transfusion there is often a great improvement in the general condition and in the anemia, but, as a rule, there is no permanent change in the temperature or the bacterial content of the blood. In discussing blood cultures, mention was made of the transient disappearance of bacteria from the blood when a reaction followed an intravenous injection.

---

[1] Severe diarrhea may occur.—Editor.

Transfusions are usually advised when the hemoglobin falls below 60 per cent. It has been found that this is about the point at which the anemia, of itself, causes symptoms. In the active stage of the disease the object is to improve the anemia and its attendant symptoms, and also to help the patient combat the infection. In the bacteria-free cases, the object is to relieve the profound anemia, and here the results are often striking. It is remarkable how the cardiac efficiency will at times be improved following a transfusion in the latter group of cases.

It is wise to put the patient on a Karell diet for twenty-four hours preceding the transfusion, or in some other way to restrict fluids in order not to overburden the heart by the additional volume of fluid injection into the blood-stream. Where edema is present it is advisable to restrict the fluids for several days before the transfusion. If the fever should be higher in the evening than in the morning, it is preferable to transfuse in the morning so that, if a reaction follows, it occurs at a time when the temperature is not high. A febrile reaction superimposed upon an already elevated temperature may be detrimental to the patient. We make it a practice to explain to the relatives that because of the presence of vegetations on the valves there is always danger of an embolism occurring and that embolism might take place during the transfusion, the procedure not being at all responsible for it. As a matter of fact, we have never had an instance of embolism during the transfusion of any of these cases.

The amount of blood given should not exceed one-fourth of the calculated volume of the blood of the patient, and generally one-eighth is sufficient. Where the myocardial symptoms are marked, the amount of blood should be still less in order not to overtax the heart. We have hitherto preferred the method of Unger or of Lindeman for the cases with high fever. On the other hand, where the fever was low or absent, we generally advised the use of citrated blood. The recent observations of Lewisohn may cause us to modify our views, as he finds that reactions more frequently follow the employment of the Unger method than the use of citrated blood.

It is important to test out the patient's blood against that of the donor before each transfusion because of the danger of hemolysis due to immune hemolysins which may develop after repeated transfusions. For other details, the reader is referred to the article of Ottenberg on Transfusions (see Chap. IX, Vol. II).

Treatment of the Myocardial Symptoms.—It has been sufficiently emphasized that, in the presence of profound anemia, cardiac efficiency is greatly depressed and, as a result, the myocardial symptoms greatly aggravated. Transfusions in such cases are sometimes attended by striking improvement of the cardiac power, and should be carried out in accordance with the principles stated above. Severe decompensation is not frequent, but, when the symptoms of myocardial insufficiency do develop, the principles underlying the therapy are those applicable to valvular or myocardial disease occurring in the absence of a superimposed bacterial infection.

General Measures and Treatment of Toxemic Symptoms.—The general measures which are useful are those employed in the treatment of any prolonged infectious disease such as tuberculosis. Absolute rest in bed should be enjoined when the fever is high. When the fever is not high, it may be of advantage to encourage the patient by having him sit up out of bed for a few hours each day, and sometimes patients are benefitted by giving them this liberty. As a result of the psychic improvement, they often take more food and digest it better. In the bacteria-free cases, the fever is usually low or absent, and the regulation of the privilege of sitting up out of bed and going out is gaged exactly as it is in other cases of cardiac disease and anemia.

When a method of therapy is being carried out which may result in any form of reaction, it is well to insist upon complete rest in bed. The diet should be non-irritating, nourishing and well prepared, and an attempt made to supplement the feeding, in order to compensate for the loss of flesh resulting from the increased metabolic rate. Bitter tonics are often useful to promote the appetite.

Little is accomplished by the use of antipyretics, but there is no objection to the use of them in small amounts in the early course of the disease. Tepid sponges will be found useful when the temperature rises above 103° F. If the patient perspires a great deal, he will find flannel night-shirts more comfortable than those made of cotton. Small doses of atropin may be tried. For the pains, phenacetin, aspirin, pyramidon, or small doses of codein may be prescribed. Morphia is rarely required. Attention should be paid to the care of the bowels. Special care should be given to the skin in order to avoid bed sores. The additional treatment of the anemia is exactly the same as in other forms of secondary anemia.

Treatment of the Embolic Phenomena.—Embolisms to the spleen and kidneys, as a rule, do not require special treatment. The cerebral embolic phenomena are generally fatal. If the patient recovers there may be a residual hemiplegia requiring treatment. Subarachnoid and intraventricular hemorrhages are not always fatal. Lumbar puncture in some of these cases may be of definite value. It is important not to remove too much fluid nor too quickly. Lumbar puncture is of value in relieving symptoms in cases in which the cerebrospinal fluid is increased, with or without the presence of polymorphonuclear leukocytes. Embolic aneurysms developing in the extremities can be treated surgically if they give marked symptoms.

Treatment of the Bacteria-free Cases.—Inasmuch as the vegetations in the bacteria-free stage are healed and the blood sterile, the measures used in the active stage to eradicate the infection obviously are not applicable here. The chief symptoms requiring therapeutic consideration are the profound anemia, the myocardial symptoms, and the nephritis. The principles underlying the treatment of the anemia have already been discussed. The anemia is closely related to the myocardial symptoms which are markedly aggravated when the anemia is severe. Mention has already been made of the striking improvement in the cardiac efficiency which results in some of the bacteria-free cases following transfusion.

What has been said of the treatment of the myocardial symptoms in the active stage also applies here. The chronic nephritis which develops in many of the bacteria-free cases is generally progressive and therapy is of little avail. The treatment is the same as that employed in azotemic nephritis due to other causes. As for the treatment of the other symptoms, the same principles should be followed as when they occurred during the active stage.

Treatment of Focal Infections.—The anhemolytic streptococci and the influenza bacillus responsible for the disease are believed to gain entrance into the blood-stream from the teeth, the tonsils, the nasopharyngeal mucous membrane, the accessory sinuses, and possibly the intestinal tract. When the bacteria are in the blood, particularly if the fever is high, nothing is gained by removing diseased tonsils or teeth unless they cause local symptoms requiring treatment. In some of the bacteria-free cases and especially in the cases of complete recovery, it is worth while to clear up foci of infection in an effort to prevent recurrences which are known to occur.

Surgical Relations.—In a patient seen for the first time or one not carefully observed, the abdominal symptoms at a given time may occupy such a prominent position in the clinical picture that an erroneous diagnosis of a surgical condition may be made, and the presence of endocarditis overlooked. In embolisms to the spleen, the pain, tenderness and rigidity over the splenic area may be so marked as to simulate abscess of the spleen. Embolism to the mesenteric arteries may cause abdominal pain with bloody diarrhea. In some cases the enlargement of the liver with tenderness and rigidity in the right upper quadrant leads to an erroneous diagnosis of liver abscess, cholecystitis, or subphrenic abscess. At times severe abdominal pain occurs without demonstrable cause, and it may occur as part of Henoch's purpura. Horder suggested the presence of small omental hemorrhages to explain some of the abdominal pains, the cause of which is not known. A knowledge of the circumstances under which abdominal pain occurs during the course of the disease will often spare the patient an unnecessary operation. We have seen at least 6 cases of the disease in which the abdominal findings were of such a character as to warrant the surgeons to operate for an acute appendicitis. In none was the appendix found to be acutely inflamed.

Infarction of the kidney not infrequently gives rise to a train of symptoms which may simulate an acute surgical condition. There may be severe pains over the kidney area, with or without radiation to the thigh or genitals, associated with nausea and vomiting, an increase in the leukocytes and a sharp rise of temperature. The finding of marked albuminuria or hematuria will often lead to the diagnosis of infarction. The bladder symptoms, frequency, tenderness, hypogastric pain, and sometimes retention, are often misleading. The incorrect interpretation of such symptoms, which result from infarction of the kidney, has occasionally led to the erroneous diagnosis of tuberculosis of the kidneys or the bladder, pyelonephritis, renal calculus or perinephric abscess. We have seen 2 cases in which a kidney was removed because of such an error in diagnosis. It is important to keep in mind that the embolisms do not lead to suppuration. For that reason one must be conservative in operating on foci in such patients, which appear clinically to be purulent in nature.

In an occasional case, the bone pains may be so severe as to simulate osteomyelitis. In one case, symptoms resembling osteomyelitis of the femur were produced by an embolic aneurysm of the femoral artery. This patient was operated upon and the aneurysm was treated by ligation. Reference has already been made to the occasionally necessary surgical treatment of embolic aneurysms occurring in the extremities and to the operative removal of focal infections.

Prophylaxis

Sir Thomas Horder was the first to emphasize the importance of prophylactic measures. Inasmuch as the great majority of cases are due to superadded infections of valves which are the seat of previous rheumatic or syphilitic disease, the broader problem becomes one of the prevention of rheumatic fever and syphilis. The more immediate consideration is the prevention of subacute bacterial endocarditis in patients who already have valvular disease. Cognizance should be taken of the fact that many of the patients who develop the disease were never aware of a previous valve lesion. In other cases, a valve lesion giving no symptoms was accidentally discovered months or years before the onset of the subacute bacterial infection. In another larger group, the patient was aware of a previous valve lesion, but the symptoms were too slight to cause him to change his habits of life. As was pointed out above, individuals showing marked symptoms of decompensation practically never develop the disease. The problem of prophylaxis should therefore be directed particularly to the cases in which the valve lesion is trifling or, at the most, of moderate severity, for these are the patients who are the most susceptible to the disease.

These individuals should keep themselves under observation. Search should be made at regular intervals for a focus of infection representing a possible portal of entry for the anhemolytic streptococci and the influenza bacilli. The resistance of the patient should be raised in every possible way. Prophylactic vaccination should be attempted on a large scale, vaccines being prepared when possible from one of the foci of infection. Otherwise mixed vaccines containing various strains of streptococci and influenza bacilli from other cases of endocarditis might be tried. In some preliminary studies it was found that such immunization gives rise to antibodies in the blood. That, of course, is not sufficient evidence that any immunity has been produced. It is only by attempts made in many places to cut down the incidence of the disease by immunization that we will learn much concerning its value. The use of non-specific protein inoculation has also been advised to increase the resistance of individuals who are the subjects of valvular disease. We have had no experience with this method.

There is no field in preventive medicine in which it is more important to carry on studies than in the prophylaxis of this disease which is practically always a secondary infection and which attacks by preference patients having valvular disease, who are still efficient.

ACUTE BACTERIAL ENDOCARDITIS

In acute bacterial endocarditis, previously diseased valves are frequently involved, but not with such regularity as in the subacute cases. The gonococcus, for instance, attacks perfectly healthy valves in the great majority of cases. On the other hand, the Streptococcus hemolyticus attacks diseased valves in about 60 per cent of the cases. The patients are generally unaware of a previous valvular lesion.

Etiology and Pathology.—The organisms responsible for the disease are nearly always the pyogenic bacteria, among which are the Streptococcus hemolyticus, the Staphylococcus aureus and albus, the pneumococcus, gonococcus, meningococcus and other Gram-negative cocci, Bacillus anthracis, etc. The vegetations on the heart valve are generally quite large and have a soft friable consistence. In a few cases the vegetative lesions are surprisingly small. The greater part of the vegetative masses consists of fibrin, agglutinated blood-platelets and large numbers of bacteria. The lesions have a great tendency to cause destruction and ulceration of the valve with extension to the adjacent myocardium. In an occasional case, perforation between one cavity of the heart and another results. The vegetative as well as the metastatic lesions are of a purulent nature.

Unlike the cases of subacute bacterial endocarditis, the valves on the right side of the heart are frequently affected. The gonococcus and, to a lesser extent, the pneumococcus, has a special tendency to involve these valves. Any of the pyogenic bacteria can, however, become implanted on the valves on the right side, and not infrequently isolated involvement of either the tricuspid or pulmonary valve occurs.

In most of the cases there is some obvious focus from which the organisms have gained entry into the blood-stream, such as a furuncle or a suppurative wound, paronychia, puerperal endometritis, osteomyelitis, or gonococcic urethritis or prostatitis. In certain instances, the portal of entry is never discovered, and then the diagnosis is often difficult.

Symptoms.—The symptoms of acute bacterial endocarditis are those of the local lesion from which the organisms have gained access to the blood, and those due to the general infection caused by these organisms, both before and after they have established themselves on the heart valve; to these may be added the embolic phenomena, resulting from the liberation into the blood-stream of fragments of the vegetative masses. The symptoms due to the general infection are too well known to require enumeration here. In a general way, they include remittent or intermittent pyrexia, rigors, sweats, severe anemia, prostration, and delirium.

There may be embolism to the various organs and the extremities. The resulting symptoms are similar to those occurring in the course of subacute bacterial endocarditis, modified by the purulent character of the infarcts produced. Frequently no symptoms referable to embolism are present.

There are instances of the disease in which the most experienced observers have failed to elicit any physical signs referable to the heart valve. The symptoms may then be exclusively those of the general infection. In such cases, as well as in those where the diagnosis is obscure, the finding of what we have termed the "Janeway lesion" is of the greatest value. These lesions, which vary in size from 0.5 to 3.0 cm., are situated on the palms of the hands and soles of the feet. They are for the most part erythematous, and partly hemorrhagic. They are generally macular, but may be slightly elevated, and have a pink or reddish color which later becomes bluish red. After a few days they disappear, leaving behind them only a slight brownish staining of the skin. In contradistinction to the Osler nodes, which are almost pathognomonic for the subacute cases, they are not tender. Janeway lesions are not present in all cases of acute bacterial endocarditis, but when they do occur they are believed to be almost pathognomonic.

Petechiæ.—Petechiæ occur often in great numbers. Purpura is not uncommon. In the staphylococcus cases, petechiæ with raised white centers are present, and constitute a characteristic finding. The elevated central portion of the petechiæ represents a minute abscess, and the surrounding redness consists of a zone of hyperemia and hemorrhage. Such petechiæ also occur in general staphylococcus infections without endocarditis.

Murmurs.—The development of fresh murmurs or of modifications of preëxisting murmurs often gives a clue to the diagnosis. The absence of murmurs does not, however, rule out the diagnosis, because not infrequently cases are encountered in which the diagnosis is verified by postmortem examination, in which no murmurs were heard during the entire course of the disease, despite the most careful examinations.

Blood cultures are generally positive, the organisms in most cases being grown with ease. The eye-grounds often show hemorrhages and, occasionally, optic neuritis. Of interest are the white spots in the retina, known as Roth's spots, which occur not infrequently in this form of endocarditis.

Course.—The course of the disease is short and rarely lasts longer than four or five weeks. Practically all the cases are fatal, but there are undoubtedly instances of recovery. Herrick recorded the cases until 1902, and since that time other reports have appeared. An analysis of these cases shows that in many no definite proof of the existence of endocarditis has been furnished. Some of them represent merely instances of a general infection without endocarditis. In the opinion of some of the best observers, there is more of a chance for recovery when the infection is due to the gonococcus than when originated by other bacteria.

Treatment.—The treatment of the disease should take into account the local lesion as well as the general infection. The surgical treatment of the suppurative focus, representing the portal of entry, may be attempted, but is generally of no avail inasmuch as the bacteria are being distributed to the blood-stream in large numbers by the vegetations. A host of antiseptic drugs have been used in an effort to eradicate the infection. Some of these have been mentioned when the treatment of subacute bacterial endocarditis was discussed. None of them has been proved to have any curative value. Two remedies are worth trying. One is sodium cacodylate administered, as described by Capps, in the subacute cases. Thompson and others record cases of recovery following the use of vaccines. Every type of vaccine and serum has been employed in the treatment of this disease. In the experience of many observers and ourselves, there has been no successful result with the use of vaccines, and we believe them to be of no value.

The general measures which are useful are those employed in general infections without endocarditis. Transfusions, though themselves not curative, may be tried to improve the patient's general condition while some systematic form of therapy of possible curative value is being employed. They are not even of any temporary value in very severe cases. The preventive treatment lies in the prompt care of local infections.

CHAPTER X

ACUTE MYOCARDITIS AND THE HEART IN ACUTE INFECTIOUS DISEASES

Henry A. Christian

Introduction.—Acute myocarditis and the heart in acute infectious diseases will be discussed together as to treatment because of the similarity in management, and also because the term, "acute myocarditis," is very indefinite in usage, being by some limited to a definite acute inflammatory process involving primarily the heart muscle, while others use it to express the circulatory disturbances occurring with the failing circulation of acute infectious diseases. Of these two uses of the term, the former would make it an extremely rare condition, while the latter would make it a condition of great frequency.

Acute myocarditis in the sense of a primary acute inflammation of the heart muscle is a possibility, but except for its occurrence by extension from adjacent structures as the pericardium, valves, mediastinum or lungs, accompanying cardiac infarction, or as part of a pyemia, it is of extreme rarity. Such primary cases have been reported but the writer has never encountered one either in the clinic or postmortem room; few, if any, of the reported cases have had an actual diagnosis made during life. When occurring with these other conditions the acute myocarditis is completely overshadowed.

Degenerative processes, fatty, hyaline, hydropic, granular or so-called cloudy swelling, occur often in patients dying from acute infectious diseases, but just what part these anatomical changes play in the circulatory failure, it is difficult to state. Furthermore, the matter is again complicated by our lack of knowledge of the parts played individually by the heart muscle and by the peripheral circulation in causing circulatory failure in infectious diseases. The view of Romberg and his associates that the circulatory failure is all a matter of vasomotor change has had doubt thrown on it by the experiments of Newburgh in the laboratory of W. T. Porter, who found the vasomotor mechanism intact in animals dying of experimental pneumonia. However, circulatory failure, whatever its mechanism, is a definite clinical entity in infectious diseases and one of very great importance in relation to prognosis in these conditions.

The acute myocardial changes of rheumatism have a much more definite basis than those of other acute infectious diseases in the presence of the Aschoff bodies scattered through the myocardium as well as degenerative changes. Usually there are combined with the muscle changes lesions of valves and pericardium. In most cases of rheumatism the myocardial lesions are of relatively little import in the acute stages of the disease, but play a large part in determining the subsequent course of life after chronic valve lesions have developed. Rarely they cause acute circulatory failure when endocarditis and pericarditis are absent.

Symptomatology.—Judging from a few reported cases of primary acute myocarditis, and from both the descriptions of others and my own observations of circulatory failure in acute infectious diseases, the symptomatology is very different from that seen in the decompensation of cardiac valvular disease and chronic myocarditis. In the first place, pallor, often of an ashen gray appearance, occurs rather than cyanosis. Edema of the dependent parts almost never occurs. Pulmonary edema may be present. Hepatic congestion with tenderness in the liver region is more frequent. The pulse is small, weak, of low tension and thready; sometimes it is rapid, sometimes slow. Arhythmia is common; extrasystoles, auricular fibrillation or flutter and partial or complete block all occur. In cases with sudden death ventricular fibrillation seems not improbable but we have no positive knowledge of this. The patients usually are in a cold sweat and nausea and vomiting are frequent symptoms.

Cardiac or circulatory failure is a serious complication of diphtheria (Place). Coming here, as it does often after the process in the throat has cleared, it gives a cleaner-cut clinical picture than in those acute infectious diseases like pneumonia and typhoid in which the fever and toxic state of the disease are still present. In diphtheria the onset is usually sudden with weakness, restlessness, pallor and vomiting. Often epigastric or hepatic tenderness is found. The pulse usually is irregular (McCulloch) and is weak and thready. The blood-pressure is low. With heart-block the pulse is slow, 30 to 40; without it, it is rapid, 110 to 140. The heart sounds are weak with a gallop rhythm. A mitral systolic murmur may develop and the heart is moderately enlarged to percussion. If the patient recovers, cardiac instability as to rate and vasomotor instability (feeling faint on slight exertion) may persist for some time. These changes, too, may be present before cardiac failure appears and suggest that it is impending.

In typhoid fever and pneumonia circulatory failure occurs much more in the febrile period of the disease. In pneumonia the cyanosis from the pulmonary process complicates the picture, and in both diseases the patient's general poor condition and the toxic manifestations of the underlying diseases obscure to a large extent the picture of heart disturbance, except the peripheral manifestations in the pulse. This also applies to the circulatory failure in the other acute infectious diseases. In these, arhythmias, such as auricular fibrillation and heart-block, may develop suddenly. In typhoid fever, sometimes during convalescence, particularly as a result of sitting up too soon or other exertion, the pulse rate increases, an apex systolic murmur appears and the heart enlarges to percussion. Return to rest ordinarily ends such a disturbance in a short time.

Treatment.—*Rest.*—Rest is the most important feature of the treatment of acute circulatory failure. A light ice-bag over the heart is indicated. The patient is to be spared all possible body exertions. The diet is to be light. In typhoid fever, where the fluid intake may have been large, this is to be reduced to a 1,200 c.c. to 1,500 c.c. level. Small doses of morphin are very helpful when the patient is restless. Of course, the infectious disease of which the circulatory failure is a complication should have the best possible treatment. If the patient recovers, a long period of rest in bed and a slow return to activity is indicated.

Digitalis.—First of all, it should be emphasized that digitalis does little or no good in these forms of acute myocarditis and circulatory failure in acute infectious diseases with a previously normal heart, except in the occasional case that develops auricular fibrillation or other similar arhythmia. These are strong statements but my own observations are not confirmatory of belief in any value of digitalis for such patients, with regular rhythm, and I find many excellent clinicians take the same view. Even with fibrillation many are skeptical of the value of digitalis. However, in the event of fibrillation developing I would at once give digitalis in large doses by mouth or strophanthin or ouabain intravenously (described in Chapter XI.) Rarely these drugs seem to be effective when fibrillation is not present. Whether digitalis is harmful to these patients when not fibrillating is still under discussion. Even if it is not harmful, its usual failure to improve the circulation is sufficient reason for not employing it routinely in the treatment of acute circulatory failure.

Will the routine use of digitalis prior to the development of signs of circulatory failure prevent their occurrence? Many believe that it will. I myself doubt it. It does not seem to me that the evidence in favor of such prophylactic use of digitalis is at all convincing. More observations are needed. To be of real value a large number of patients must be observed with alternate cases not receiving digitalis serving as controls. Digitalis has been advocated most as a routine in the treatment of pneumonia. One reason for this was to have the patient well digitalized so that, when any circulatory disturbance came, a quick digitalis effect might be obtained. This reason, I think, no longer holds because by the large dose method of giving digitalis by mouth or by the intravenous use of strophanthin or ouabain (described in Chapter XI) equally prompt digitalis effects are attainable. I do not employ digitalis at present as a part of the routine management of pneumonia because my own use of it has not seemed to indicate any beneficial effect.

Nothing of what has been said of digitalis applies to those patients who have evidences of cardiac decompensation from some preëxisting cardiac lesion. For these I would use digitalis from the beginning of their acute illness as described in Chapter XI, the method of giving and the dosage depending on the evidences present of cardiac decompensation.

Quinidin Sulphate.—Quinidin sulphate may prove beneficial in those cases developing auricular or ventricular fibrillation or numerous extrasystoles. (For discussion of quinidin see Chapter XI.) So far this is more hypothetical than proved by observation.

Cardiac Stimulants.—Are there any? I doubt it. Digitalis is not a cardiac stimulant in this type of cardiac failure. Strychnia has a temporary improving effect on the pulse and for its use there is no contraindication. The consensus of opinion, however, is against its being of much real value (Parkinson and Rowland). The same applies to camphor given subcutaneously in combination with ether and olive oil (camphor 1.5 gm., olive oil 6 c.c., ether 4 c.c.; dose 1 to 2 c.c. subcutaneously). Caffein, 0.1 to 0.3 gm. dose, given every four hours, occasionally seems to have some effect in improving the circulation. All of these appear to have more influence on the peripheral circulation than on the heart. Restraint in medicinal therapeutics seems the wisest procedure. In the face of marked circulatory failure in acute infectious conditions, I would use caffein in combination with either strychnia or camphor, caffein 0.1 gm. (1½ gr.) and strychnia 1½ mg. (1/40 gr.) subcutaneously every two hours for a maximum of six doses; occasionally they seem to tide over the emergency; often they cause no improvement. The routine use of such drugs in lesser degrees of circulatory failure is undesirable.

REFERENCES[1]

Aschoff and Tawara. Die heutige Lehre von den pathologisch-anatomischen Grundlagen der Herzschwäche, Gustav Fischer, Jena, 1906.

Coombs. Rheumatic Myocarditis, Quart. Journ. Med., ii, 26, 1908-1909.

Holst. Acute Myocarditis, Nelson Loose-Leaf Medicine, iv, 386, Thomas Nelson & Sons, New York, 1920.

McCulloch. Studies on the Effect of Diphtheria on Heart, Am. Journ. Dis. Child., xx, 89, 1920.

Newburgh. The Vasomotor Mechanism in Pneumonia, Am. Journ. Med. Sc., cxlix, 204, 1915.

Parkinson and Rowland. Strychnia in Heart Failure, Quart. Journ. Med., vii, 42, 1913-1914.

---

[1] For papers on digitalis and quinidin see references to Chapter XI.

Place. Diphtheria, Oxford Medicine, v, 147. Oxford University Press, American Branch, New York, 1921.

Porter and Newburgh. The State of the Vasomotor Apparatus in Pneumonia, Am. Journ. Physiol., xxxv, 1, 1914.

Romberg, Pässler, Bruhns, and Müller. Experimentelle Untersuchungen über die allgemeine Pathologie der Kreislaufstörung bei acuten Infectionskrankheiten, Arch. f. klin. Med., lxiv, 652, 1899.

CHAPTER XI

TREATMENT OF CHRONIC MYOCARDITIS AND CHRONIC VALVULAR DISEASE OF THE HEART

HENRY A. CHRISTIAN

Introduction.—From the point of view of treatment, chronic myocarditis and chronic valvular disease of the heart can be discussed advantageously together for two reasons: (1) because primarily the symptoms and other disturbances in each depend on the condition (functional efficiency) of the heart muscle, and (2) because the same principles of treatment apply for each condition. A few minor variations in management, dependent on the particular lesion, will be discussed after the general principles of treatment applicable to the vast majority of cases in these two groups have been described. What we are called upon to treat under the heading of this chapter really is chronic heart disease or chronic cardiac decompensation, and these terms will be used in referring to cardiac cases with chronic disturbances of valves or muscle, or, as is usually true of cases with chronic valve lesions, with chronic disturbances of both valves and muscle.

Three factors may enter into the production of the symptoms and disturbances resulting from chronic heart disease: (1) the muscle is, in itself, inefficient in its function; (2) valve lesions cause mechanical hindrance to muscle effectiveness; (3) arhythmia disturbs the orderly sequence of muscle contractions and so hinders their efficient action. Indirectly, increased peripheral resistance is a factor in causing cardiac inefficiency. Of these, in relative importance, muscle comes first, and the mechanical effect of valve lesion comes last.

PATHOLOGICAL PHYSIOLOGY

In *chronic myocarditis* a diseased condition of the heart muscle interferes with an adequate systolic output of blood per unit of time. So long as the systolic output is maintained at such a level as to furnish to the body the needed blood supply, there are no disturbances in heart function; when systolic output decreases below this level, symptoms begin to appear. Systolic output may, and very frequently does, decrease without change in rhythm, sometimes even without increase in rate. Each systole of the heart is less powerful, less complete or comes when the ventricles are less completely filled than is normal. Any or all of these may happen without any disturbance in rhythm or any valve defect, and be dependent on some lesion which damages the heart muscle. In addition, arhythmias may so disturb the orderly sequence of muscle contraction that there are so many systoles coming after a very brief period of diastolic filling of the ventricles as to result in a decreased systolic output and so decreased cardiac function; or conduction disturbances may so slow heart action as to give a systolic output inadequate for normal body function. Arhythmias usually occur in hearts with diseased myocardium and add their effect to those coming from the diseased heart muscle with regular rhythm. Occasionally the arhythmia is the sole disturbing factor.

In chronic myocarditis it is the musculature of the ventricles that is chiefly concerned; with regular rhythm the condition of the musculature of the auricles is of very little importance. However, muscle lesions in the auricles may cause disturbances in rhythm and indirectly affect cardiac action by disturbing ventricular function and so decrease the systolic output of blood.

For these disturbances in heart muscle we use the term "chronic myocarditis." However, the term is definitely unsatisfactory. It means inflammation and implies connective tissue increase. In many of these hearts there is no evidence of inflammation, and there is no increase in interstitial connective tissue. Sometimes we find evidence under the microscope of degenerative changes in the heart muscle but very, very often the miscroscope fails to reveal any lesion of the heart muscle. This is not the place to discuss the various theories as to just what has happened in these hearts to disturb muscle function so greatly. They are interesting theories, but they fail to give any really satisfactory explanation of the mechanism of muscle failure in these patients. During life we cannot anticipate which heart will show connective tissue increase, which heart will show degenerative changes in heart muscle, or which heart will show under the microscope essentially normal appearing musculature. So we use the term "chronic myocarditis" to include all, and to mean a heart in which the muscle is functionally incompetent; the term is really used in a functional rather than structural sense. Other terms have been suggested but they are as little satisfactory.

In chronic myocarditis the prime disturbance is the ineffectual contraction and decreased tonicity of the muscle. In addition arhythmias often add another disturbing factor. Organic valve lesions are lacking. However, valve orifices become enlarged as a result of muscle disturbances and regurgitation through these dilated orifices is a further factor disturbing heart function. This sort of regurgitation may take place at any valve, but it is relatively an infrequent occurrence at the pulmonic and aortic valves [1] and of very little consequence to heart function when occurring. On the other hand it is commonly present at the mitral and tricuspid valves and plays an important part in cardiac inefficiency, even though it is a secondary result of myocardial disturbance.

In chronic myocarditis the heart, with rare exceptions, is enlarged; when the condition produces symptoms, the heart is almost always very considerably enlarged; so much so that it is usually easy to detect enlargement by simple physical examination, palpation, percussion and auscultation. Only rarely is an X-ray needed to determine the enlargement in these cases; usually those that require X-ray are patients with marked pulmonary emphysema in whom the air-distended lung covers over the heart with so thick and resonant a layer as to interfere with palpation, percussion and auscultation. My own feeling is that X-ray very infrequently is of any essential value in the study of chronic cardiac cases, the exception being the markedly emphysematous lung or the occasional case when it may be needed to distinguish displacement from enlargement. Cardiac enlargement in these patients is due to thickening of the heart wall and enlargement of the cardiac cavities (hypertrophy and dilatation). Apart from hypertrophy, dilatation, though much talked about, in my judgment, plays but an insignificant rôle in chronic cardiac failure. It is a very striking thing that, as a rule, these enlarged hearts show no demonstrable decrease in size as the result of effective treatment, and it would seem that once an enlarged heart, always an enlarged heart, is true except for the slighter changes in size that occur as part of the physiologic variation of cardiac function. Genuine acute dilatation practically does not occur so far as my experience goes.

Chronic endocarditis leads to thickening, contraction, interadherence, and retraction of the valve cusps. The chordæ tendineæ usually are involved too with thickening and shortening. The result is either a narrowing of the valve orifice or a failure of the cusps to close the orifice. Very frequently both occur and there are signs of stenosis and insufficiency. The recognition of the various valve lesions depends on simple physical examination and needs no description here. In these valvular lesions almost always what has caused the damage to the valves simultaneously has injured the heart muscle, or the stress and strain of the muscle struggling against the mechanical defect of valve lesion has caused damage to the muscle; as a result chronic myocarditis, in the sense already explained, is a frequent accompaniment of the chronic valve lesion and is the important factor in producing symptoms and other disturbances. In many patients it is the chronic myocarditis that is the chief factor in causing cardiac decompensation, as shown by the response to therapeutic measures that can have no effect on the valve lesion. However, in these cases the mechanical effect of the valve lesion is always some factor in the disturbance and sometimes, as in marked aortic regurgitation and in extremely stenosed valve orifices, it is the chief factor. This is to be borne in mind when evaluating the probable effect of therapeutic measures.

---

[1] Relative insufficiency of the aortic valves is probably more common than has usually been taught.—Editor.

The *arhythmias* that play any conspicuous part as actual hindrances to efficient muscle action and in determining symptoms and disturbances of chronic heart disease are relatively few and easily recognized by the finger and ear of the clinician who has had these sense organs trained under the check of polygraph or electrocardiograph. These instruments, after they have had their educational use, are of relatively little importance in the treatment of chronic cardiac disease.

Of these arhythmias the most important, from the point of view of both frequency and as a disturbance to cardiac function, is *auricular fibrillation,* which gives to the pulse an irregularity without any dominating rhythm, but merely a succession of impulses of varying force, varyingly placed. When auricular fibrillation is acting as a factor disturbing cardiac action, the rate is rapid, and when counted with the stethoscope over the heart, there is an excess of beats over those that can be felt by the palpating finger on the radial artery, constituting what we term a pulse deficit. These phenomena are sufficient to justify the diagnosis of auricular fibrillation with but very few exceptions. Rather closely allied to auricular fibrillation is *auricular flutter,* sometimes recognizable from fibrillation by the palpating finger because of a certain rhythmic variation in the irregularity. If unrecognized as flutter by simple palpation and auscultation, flutter would pass as fibrillation and the clinical significance is much the same. The third arhythmia of functional importance, though much more infrequent than fibrillation or even flutter, is complete *heart-block* with greatly slowed heart rate. Rates above 35 are rarely of functional significance and rates below 35 are practically always complete heart-block, and may be so diagnosed merely on the basis of this slow rate. *Extrasystoles* are very frequently met with in chronic heart disease and are usually easily diagnosed by the palpating finger as an occasional to frequent interruption of the regular rhythm by a beat coming at a shorter than normal interval after a regular contraction, and often followed by a recognizably longer pause than the regular interval between beats. Unless the extrasystoles are extremely numerous they probably play no part in decreasing cardiac efficiency, though they may be productive of annoying symptoms. When they recur regularly after each normal systole they constitute what is termed a bigeminal pulse. Some of the rare arhythmias play a part in decreasing cardiac function, but they are too infrequent to be of any considerable importance in the treatment of chronic cardiac disease.

Though not strictly an arhythmia, pulsus alternans is a very significant change in the pulse, indicative of a poorly functioning myocardium. In pulsus alternans the size or volume of the pulse waves varies so that every other one is smaller, indicative of a failure of the heart muscle to recover completely from the effects of one systole before the next comes. Often this rhythmic variation in pulse size can be detected by the palpating finger on the radial artery. Very frequently, when not made out in this way, the variation is apparent in taking a blood-pressure reading with a sphygmomanometer, particularly when the auscultatory method is used. However, in many cases an arteriogram is necessary to detect pulsus alternans.

When the blood-pressure is high, often alternation can be made out while registering the blood-pressure with the sphygmomanometer, though the arteriogram fails to show any variation in size of the pulse waves or a graphic record is very difficult to obtain. Not infrequently pulsus alternans, clearly recognizable by the palpating finger, may be very difficult of graphic recording. All three methods of observation should be used in order not to overlook the existence of pulsus alternans because its presence is of such importance in prognosis. A lesser degree of pulsus alternans is present in those patients in whom it can be detected only in a few beats next succeeding the pause after an extrasystole. Here it is of less serious prognostic significance than when continuously present. Variation in the size of pulse waves in auricular fibrillation, extrasystoles and other irregularities of rhythm do not constitute pulsus alternans. With *pulsus bigeminus* due to regularly recurring extrasystoles, the pulse waves alternate in size since the extrasystoles give a smaller systolic output than the regular systoles. This constitutes a false or pseudo pulsus alternans, easily recognized by the palpating finger and of different significance from true pulsus alternans. True pulsus alternans is of very great significance in prognosis; with very rare exceptions it is of grave omen. The pulsus alternans present during paroxysmal tachycardia that disappears with restoration of normal rate is one of these exceptions.

Another disturbance in heart action, not an arhythmia, is of much significance. It can be recognized only with the electrocardiograph. It consists of a distortion of the ventricular complex in the electrocardiogram with prolongation of the Q.R.S. interval and notching or thickening of the up or down strokes of the R.S. wave. This frequently is spoken of as a disturbance in intraventricular conduction, due to a lesion breaking continuity of conduction through the Purkinje's fibers. Whatever its actual cause, its occurrence is of serious import and to be interpreted much as pulsus alternans unless it is a transitory phenomenon and does not recur, as rarely happens.

ETIOLOGY

Chronic cardiac disease, in itself, has no specific etiology. Of the causes of chronic valvular disease rheumatic fever comes first in rank of etiological factors. Chorea and tonsillitis, both closely allied to rheumatic fever in our ideas of etiology, play an important rôle in causing chronic valvular disease; other types of infection are of minor importance. Syphilis is a frequent cause of aortic insufficiency. Strictly speaking this type of aortic insufficiency depends on changes in the aortic ring and adjacent aorta more than on a pathological process involving the valve cusps. However, the syphilitic process frequently does extend to the cusps and rarely it may begin in the cusps. Syphilis plays a negligible part with regard to the valves other than the aortic.

Chronic valvular disease with few exceptions has its inception in childhood or early adult life. Frequently, many years elapse before signs of cardiac disease appear. However, after forty-five it is rare to have the beginning of symptoms of chronic valvular disease of the heart from valve lesions. Conversely the diagnosis of chronic cardiac valvular disease should be made with great caution when the patient develops symptoms of heart disease after forty-five years of age. Syphilitic disease of the aorta may come at any age but is commonest in young adults and those of middle age, and in males much more common than in females.

The causes of chronic myocarditis are less well understood than those of chronic cardiac valvular disease. There is a growing belief that antecedent infections of various sorts play an important etiological part. These do not include rheumatic fever when we exclude cases with organic valve lesions, since it is extremely rare for this disease to cause lesions in the heart muscle without, at the same time, causing valve lesions. Yet when valves are involved, rheumatism very often attacks the myocardium, too, and there is an acute heart muscle lesion of rheumatic origin which is quite characteristic of that disease, namely, the rheumatic nodules or Aschoff bodies. Their occurrence very probably is responsible for some of the muscle change of the chronic stages of rheumatic valve lesions.

Hyperthyroidism, if persisting, usually leads to chronic myocarditis; experimentally thyroid preparations will cause myocardial lesions. This is an additional indication for prompt and thorough treatment of hyperthyroidism. Arteriosclerosis and hypertension frequently are antecedent to and important causative factors of chronic myocarditis. Arteriosclerosis without hypertension damages the heart muscle most probably by disturbing its nutrition as a result of coronary involvement. Hypertension, with or without arteriosclerosis, adds a factor of strain from peripheral resistance. With both there may be some direct action on the heart muscle of those causes, whatever they may be, which have produced arteriosclersis and hypertension. Curiously enough, syphilis seems to play a very minor rôle in causing chronic myocarditis.[2]

In contrast to chronic valvular disease, the symptoms from chronic myocarditis rarely occur before forty-five years of age, and if the patient is over forty-five when symptoms appear the chances of its being chronic myocarditis rather than a valvular lesion are very great.

SYMPTOMATOLOGY

The factors discussed so far, which play parts in causing insufficiency in chronic cardiac disease by decreasing systolic output below that sufficient to maintain normal body function, produce the symptoms and other disturbances which should be treated. These symptoms and disturbances are breathlessness, cough, edema (subcutaneous tissues, body cavities, viscera), cyanosis, chronic passive congestion, pain, substernal pressure, hyperesthesia, restlessness, palpitation, exhaustion and tachycardia. The most important of these as indications for treatment, and those whose amelioration shows when treatment is adequate, are breathlessness, edema and cyanosis. Occasionally chronic passive congestion of the liver, shown by enlargement and tenderness, is the chief index of cardiac insufficiency. It is important to recall that serious cardiac decompensation may exist in the absence of tachycardia.

TREATMENT

Treatment of chronic cardiac disease will be discussed under five headings: (1) treatment during the stage of heart failure or decompensation; (2) continuation treatment after the symptoms of heart failure or decompensation have ameliorated if not entirely disappeared; (3) management during the period when, though there are physical signs of cardiac disease, symptoms are absent or slight; (4) prevention of the development of cardiac disease; (5) treatment of special conditions occurring during chronic cardiac disease.

Treatment During the Stage of Heart Failure or Decompensation

By heart failure or decompensation is meant that stage in the progression of cardiac disease at which the symptoms and disturbances of the condition are sufficiently evident to constitute a definite handicap to the individual's efficiency. To put it another way, they are sufficiently marked to cause the patient to consult a physician. It is quite evident that the terms, heart failure or decompensation, are only relative. The cardiac lesion which would greatly handicap the laborer who digs ditches might produce almost no symptoms in the man whose occupation is largely sedentary; with the same actual physical condition the latter might regard himself as normal, while the former is to himself very evidently ill. This relationship must be kept in mind in planning the continuation treatment of different individuals.

---

[2] Warthin, from pathological studies claims that syphilis plays an important part in the causation of myocarditis. Clinical experience does not support this view. —Editor.

The form of treatment here advised is suitable to all types of chronic cardiac disease, if decompensated. Patients with regular rhythm are just as suited for and give just as good results from digitalis therapy as those with auricular fibrillation. Chronic myocardial cases without valve lesions give as good results as those with chronic valve lesions. All types of valve lesions react well, some better than others. High blood-pressure is no contra-indication to digitalis therapy.

Let us assume the patient is breathless to the stage of orthopnea and has evident edema as well as some of the other symptoms and disturbances of chronic cardiac disease, what are we to do to improve his condition? Treatment of such a patient resolves itself into two procedures of prime importance: rest and an increase in the effectiveness of the heart muscle in its work, mainly by the use of digitalis. In addition there are other measures of less relative importance.

Rest.—Such a patient should be put to bed comfortably. The bed should be of moderate width, not over four feet wide, so as to allow of easy access to the patient from either side by the nurse or member of the family in attendance. Incidentally, such a bed facilitates examination by the physician. The mattress should be smooth, not too soft and on springs, which, while resilient, should not sag much with the patient's weight. Wheels on the bed are helpful but not necessary. The old-fashioned double bed with soft springs or a feather tick is a very poor place for a cardiac patient.

To put the patient into a proper bed so that he will be comfortable is of next importance. Almost all cardiac patients are very uncomfortable when lying flat. Not infrequently they have been spending their time propped up in a chair. Often they are convinced that they cannot rest in bed. In my experience practically all cardiac patients can be made more comfortable in bed than in a chair, and in bed get more rest, which is an essential to good treatment of heart disease. The exception comes during paroxysms of dyspnea. For the chronic cardiac case to be comfortable in bed the patient must be propped up. Various types of bed-rest are made. To be satisfactory any bed-rest must be so arranged that the patient's buttocks are on the flat bed, and the bottom of the bed-rest begins just back of the place of contact of buttocks with the mattress. Often the mistake is made of having the patient sitting on the sloping or curved lower end of the bed-rest with a constant tendency to slip down, against which the patient is continuously exerting energy. The bed-rest that goes under the mattress is not very satisfactory because it has just such a curve at the bottom. The satisfactory bed-rest also is adjustable so as to give several angles of incline, and it is desirable to arrange the bed-rest so that there is no danger of the patient slipping off the sides while asleep. This can be accomplished by side-pieces, by a sheet rolled up and fastened on each side to the head and side of the bed, or by pillows properly arranged.

The patient on a bed-rest usually needs a support for his feet, to prevent his slipping down on the bed-rest. Either pillows or a foot stool against the foot of the bed, or a rolled-up sheet or long bolster fastened to the sides of the bed, so as to be across the bed at the level of the patient's feet, will serve this purpose.

In place of a bed-rest a number of pillows may be used. To be satisfactory they require very careful adjustment and are to be regarded more as a makeshift than a satisfactory substitute for a good bed-rest. If used it is desirable that the undermost ones be hair pillows on which feather pillows are placed. A fairly satisfactory bed-rest can be improvised with an ordinary straight-back chair, turned down, and covered with pillows.

Very satisfactory special hospital beds are made which give much comfort to the patient; the best type has two points of adjustment and a specially constructed mattress. At the head end it can be elevated to serve as a bed-rest and about halfway between the middle and foot it can be raised to give a support just under the patient's knees. This provides both an inclined plane on which to prop up the patient at any desired angle and a support to prevent the buttocks from slipping from the inclined head end. With marked edema of the legs, however, it is better to leave the lower part of the bed horizontal or very slightly elevated.

A bar, stirrup or ring suspended from the ceiling over the bed at a level easily reached by the patient is helpful in enabling the patient to move and readjust himself with a minimum of exertion. A bed table, so adjusted that the table top comes just in front of the patient at about the level of the lower end of the sternum, serves not only as a convenient place for the patient's food tray, books, etc., but is useful for the patient to lean forward on, pillows being placed on the table. To some cardiac patients this position proves very comfortable, and to many it gives a pleasant means of change of position. Obviously such a table needs to be solidly supported to prevent being tipped.

The reclining patient needs a shoulder blanket, not too large, its thickness depending on the weather. A two-piece garment, such as pajamas, should be worn by both women and men. In colder weather this should be of fairly warm material. Undershirt and drawers or union suits should not be used under the pajamas on account of the exertion incident to their change. The long nightdress, ordinarily favored by women, is not suitable to the cardiac patient on account of the exertion required to get it pulled down properly under the patient. A dainty bed jacket over the pajamas will serve to furnish the desired feminine background for female patients.

With these various arrangements made, it must be impressed on both patient and attendants that their reason is to give rest and the patient must be kept from moving so far as is possible. Bed-pan and urinal are to be used in bed. The patient is to be bathed in bed and there brush his teeth, etc. The patient is to be told not to sit forward to greet friends and when changing his position to move slowly and allow himself to be helped by others. Caution against much talking is to be given and only brief visits from outsiders are to be allowed. The well-meaning but long-winded and indiscreet friend is to be denied admittance as if he or she were a plague.

Having made the patient comfortable in bed, an all-important therapeutic procedure, especially in the evening, for the restless, sleepless, wearied cardiac is morphia subcutaneously in a dose of 15 mg. (¼ gr.) and repeat the dose later in the night if needed. This procedure should be repeated, for most patients, the second night, after which time other therapeutic measures, if successful, will render this drug no longer needed.

Digitalis.[3]—The sheet-anchor of treatment for chronic cardiac disease is digitalis, which is to be given promptly, in adequate dosage, and in the form of a potent preparation. Except for morphia, used as already described, this is the only drug required by most cardiac cases.

Preparations.—In my opinion the only essential in the selection of a digitalis preparation is that it should be potent and that the physician should know its strength in terms of its efficiency. Therapeutically, it is quite immaterial whether infusion, tincture of powdered leaves, the simple pharmacopeial preparations or those prepared specially by some manufacturing pharmaceutical firm, under such names as digifolin, digipuratum, digitan, crystalline digitoxin, digitalin cristallizée Nativelle is used. My belief is that all have the same pharmacological action and are interchangeable as to use. I do not believe that, if given in the same dosage measured in physiological effect, the one can be distinguished from the other so far as effect of any sort is concerned. This being true it is reasonable to use the preparations that are least costly. At the time this is being written prices are quoted for 100 average therapeutically equivalent doses as follows: pills of powdered digitalis leaves $2.11, digipuratum tablets $4.41, digitan (ampules of digipuratum) $11.00, Nativelle's crystals $4.37.

---

[3] A very complete review of digitalis therapy will be found in Medicine, 1922, Vol. I, p. 1, by Dr. G. Canby Robinson.

There is, therefore, very definite economy in using powdered digitalis leaves.

It has always seemed to me to be good therapeutics for a physician to use continuously one form of digitalis so as to become thoroughly familiar with its dosage and effects. Personally I have used the simplest possible preparation of digitalis, namely, powdered digitalis leaves made into pills, and had the greatest satisfaction with the results (Figs. 1 to 9). The powdered digitalis is mixed with enough very thick glucose syrup, slightly warmed, to make a sticky mass, and from this pills are rolled. Lycopodium powder is used to prevent them from sticking together. These pills should be made up at frequent intervals. If they do not crush easily between the fingers they should not be used. Starting with a potent digitalis leaf I have found such a simple preparation completely satisfactory for oral administration, and it is rare that any other method of giving digitalis is needed. All digitalis preparations should be standardized either on animals, using the frog method or the cat method, or on patients, finding out by using a given preparation what its potency is and so the proper dosage. I may add in justice to the American pharmacists that a ten years' experience at the Peter Bent Brigham Hospital has never shown an inefficient digitalis leaf, our simple pills have proved entirely satisfactory, and a recent study of digitalis preparations indicates that there has been a marked improvement in their quality in the last decade as supplied by American druggists.

Action.—In 1785 Withering described the goal of digitalis dosage in these words:

> "Let the medicine be continued until it either acts on the kidneys, the stomach, the pulse or the bowels; let it be stopped upon the first appearance of any one of these effects."

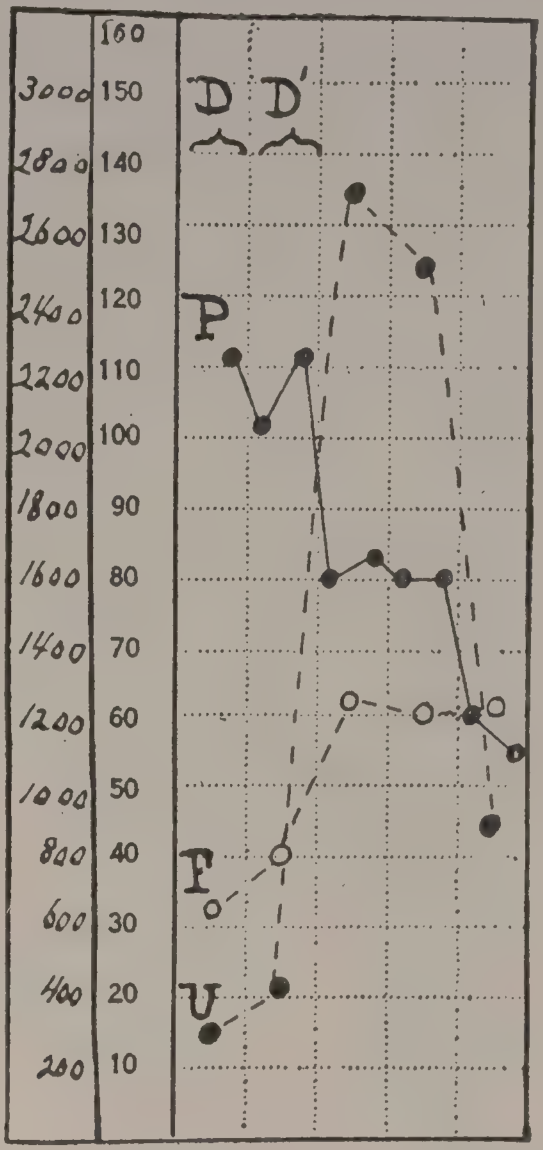


Fig. 1.—Male, Age 28, Chronic Cardiac Valvular Disease, Mitral Stenosis; Rhythm Regular. The first column of figures indicates the amount of urine output and the fluid intake for each 24 hours expressed in c.c. The second column of figures indicates the apex and radial pulse rates per minute. D = 3 doses of 0.2 gm. each of powdered digitalis leaves, a total of 0.6 gm. on this day. D^1 = 7 doses of 0.3 gm. each of powdered digitalis leaves a total of 2.1 gm. on this day. Total D + D^1 = 2.7 gm. of powdered digitalis leaves. P = pulse rate counted at the wrist. F = fluid intake measured in c.c. U = urine measured in c.c. The effect of digitalis in this case was a slowed pulse (110 to 55) and on two days a marked diuresis, with urine increase from 400 to 2,700 and 2,500 c.c.

No better criteria of dosage can be formulated to-day than given in these words of Withering. Many failures in digitalis therapy are due to the fact that physicians so often fail to continue it "until it either acts on the kidneys, the stomach, the pulse or the bowels." Withering implies that the physician should know how digitalis acts; for him this knowledge was limited to the result, while we of to-day should understand, too, the mechanism, in part, if not in its entirety.

Digitalis acts on the cardiac mechanism in several ways to slow rate (Figs. 1, 2, 3, 4 and 5) and increase systolic output. In addition there are some effects of digitalis that decrease cardiac efficiency. Fortunately these come later and may be avoided by avoidance of too large a dosage. Digitalis stimulates the vagus nerve to increase its inhibitory action, apparently acting on the central nucleus of the vagus. As a result the rate is slowed from the sino-auricular node. The vagus also influences the auriculo-ventricular or His muscle bundle to slow conduction time from auricle to ventricle. Digitalis acts, too, directly on this muscle bundle to slow conduction. Digitalis has a direct action on ventricular muscle to increase systolic output. Of this last action of digitalis there has been considerable uncertainty, but it seems to me that the evidence, particularly evidence of its action in man, leaves no doubt. The changes in the T wave of the electrocardiogram, as a result of digitalis, indicates an effect on ventricular muscle. The appearance of ventric-

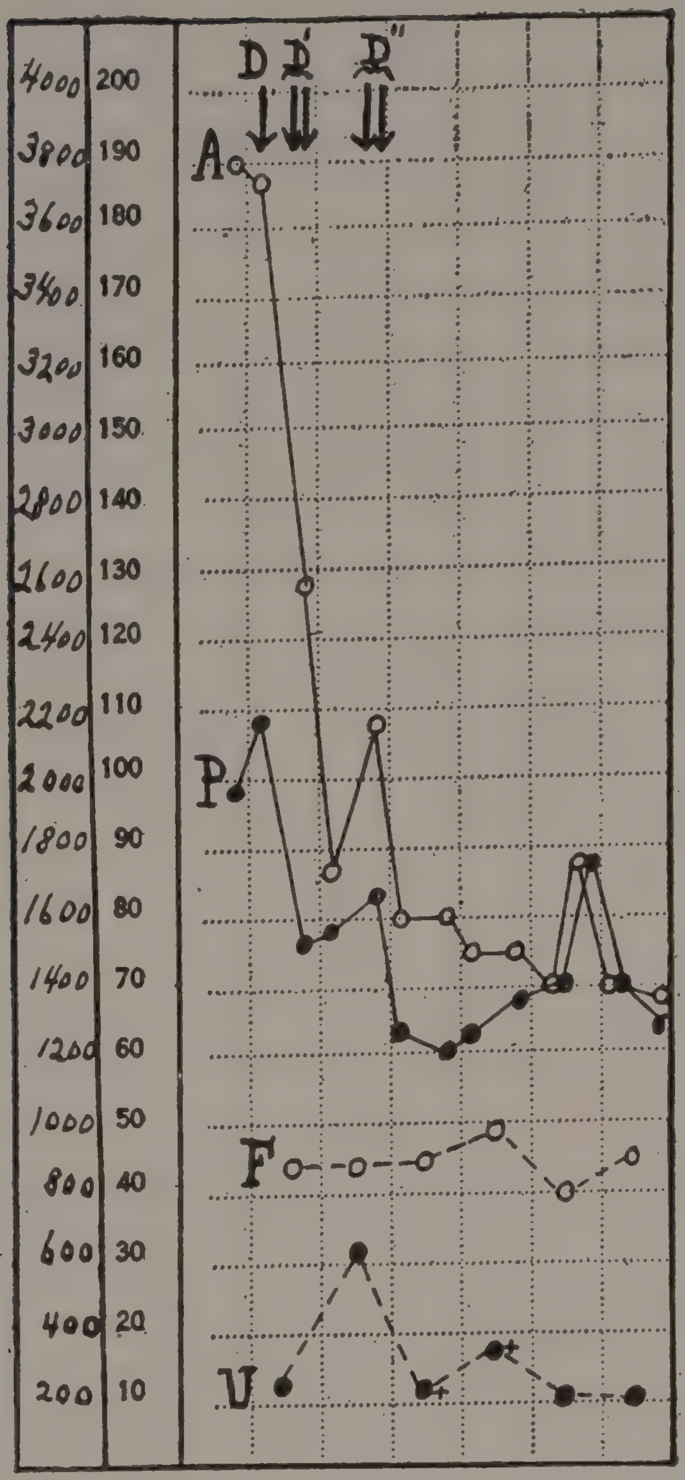


Fig. 2.—Female, Age 28, Chronic Cardiac Valvular Disease, Mitral Stenosis and Regurgitation, Aortic Regurgitation; Auricular Fibrillation. See Fig. 1 for explanation of figures on side. D = 1 c.c. tincture of digitalis given intravenously at 10.12 A. M. $D^1 = 2$ doses of 0.5 gm. of powdered digitalis leaves given at 1.52 and 8 P. M., a total of 1 gm. $D^2 = 2$ doses of 0.1 gm. of powdered digitalis leaves given at 6 and 10 P. M., a total of 0.2 gm. Total $D + D^1 + D^2 = 1$ c.c. of tincture of digitalis intravenously and 1.2 gm. of powdered leaves by mouth. A = heart rate counted with a stethoscope over the apex region. P = pulse rate counted at the wrist. F = fluid intake measured in c.c. U = urine measured in c.c. The effect of digitalis in this case was a slowed apex rate (190 to 70), with disappearance of pulse deficit.

ular extrasystoles from larger doses is another evidence. Most significant is the improvement in cardiac cases with slow normal rhythm in which rate is slightly, if at all, slowed by digitalis at a time when cardiac action is obviously improving. It is possible that digitalis acts, also, indirectly on ventricular muscle by improving nutrition as a result of improved circulation through the coronaries.

As far as changes in the heart, made out by physical examination, are concerned, the evidence of digitalis effect comes mainly in a slowed rate (Figs. 1 to 5) in cases in which the rate is rapid before treatment. The importance of judging the rate by counting the apex rate needs emphasis. The pulse may be slow because many pulse waves do not reach the wrist. In this event the radial pulse gives no index of heart rate. An irregular rhythm is very rarely rendered regular by digitalis except in auricular fibrillation when a complete auriculoventricular block converts an absolutely irregular pulse into a slow and regular one, and in auricular flutter when digitalis leads to reëstablishment of the normal rhythm. As digitalis increases the

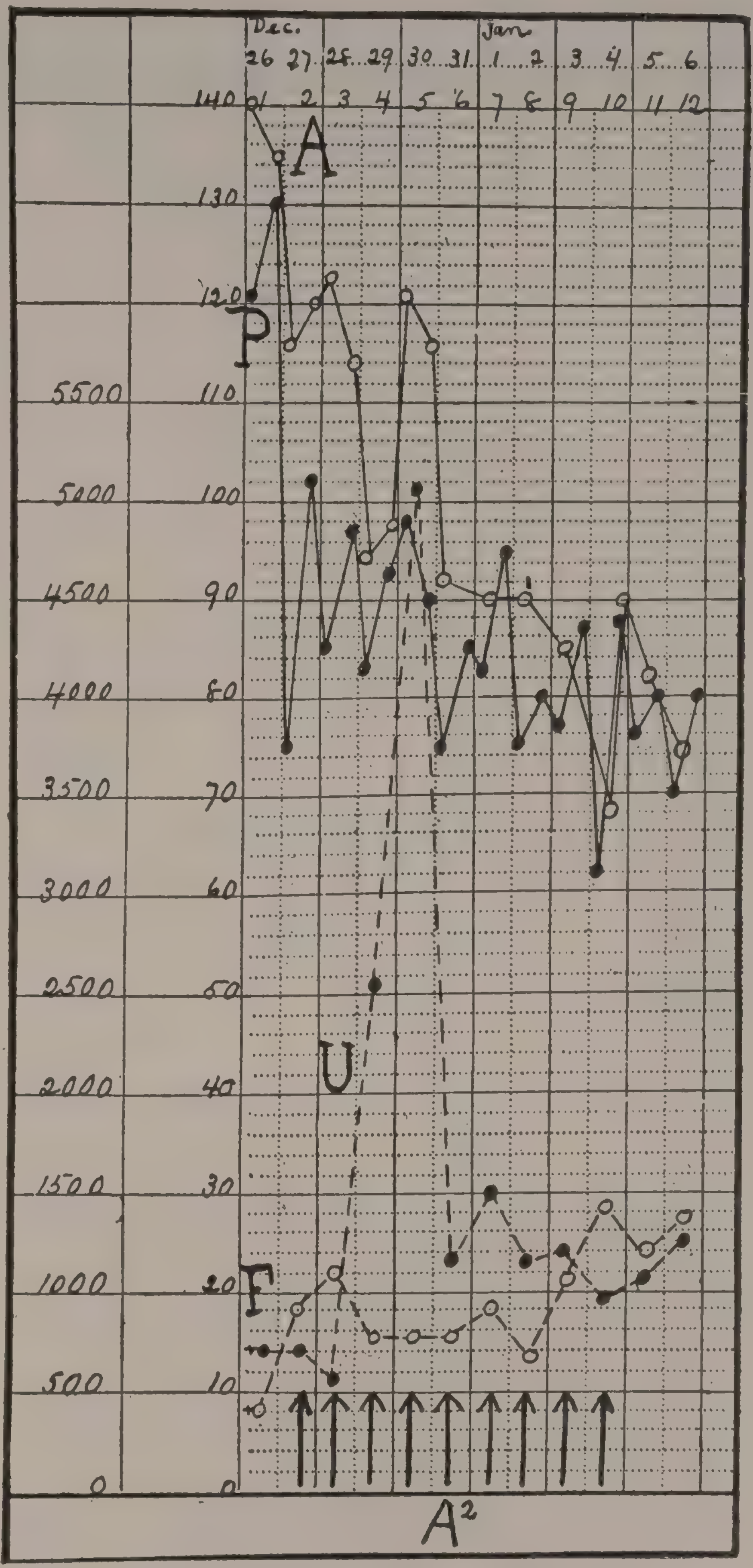


FIG. 3.—MALE, AGE 57, CHRONIC MYOCARDITIS, AURICULAR FIBRILLATION. The first column indicates the urine output and fluid intake for each 24 hours expressed in c.c. The second column indicates the apex and pulse rates per minute. The arrows at A² indicate days on which the patient received three doses of 0.1 gm. each of powdered digitalis leaves. A = heart rate counted with a stethoscope over the apex region. P = pulse rate counted at the wrist. F = fluid intake measured in c.c. U = urine measured in c.c. The effect of digitalis in this case was a marked slowing of the heart rate from 140 to 74 and a urine increase from 550 to 2,525 and 5,550 c.c. per 24 hours.

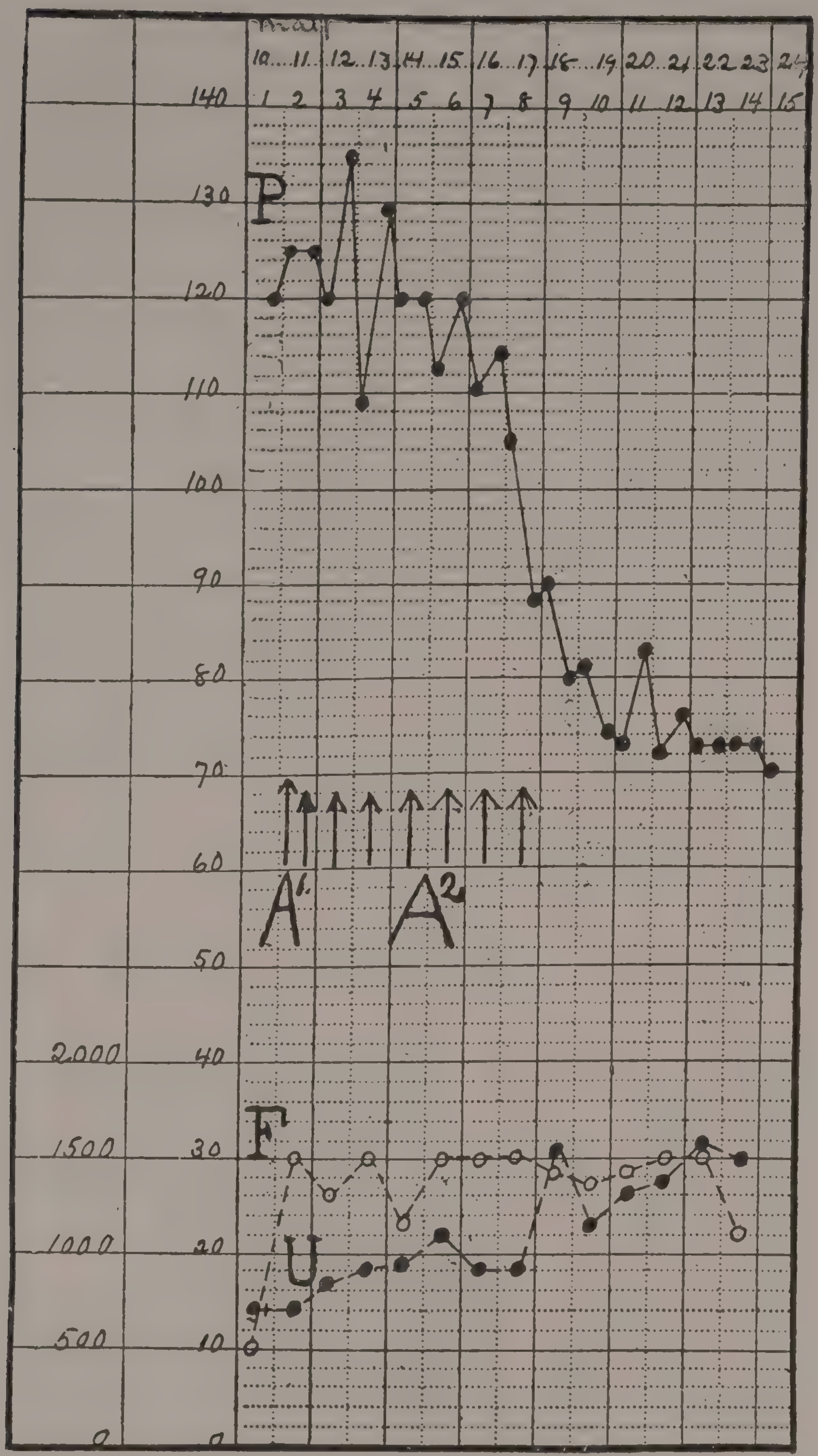


FIG. 4.—MALE, AGE 35, CHRONIC MYOCARDITIS, REGULAR RHYTHM AND MARKED DEGREE OF PULSUS ALTERNANS. The first column of figures on the left hand side of the chart indicates the amount of urine output and fluid intake for each 24 hours expressed in c.c. The second column of figures on the left of the chart indicates the apex and pulse rates per minute. Arrow over A¹ indicates intramuscular dose of 1 c.c. of digipuratum. Arrows over A² indicate days on which the patient received three doses of 0.1 gm. each of powdered digitalis leaves. P = pulse rate counted at the wrist. F = fluid intake measured in c.c. U = urine measured in c.c. The effect of digitalis in this case was a marked slowing of the pulse from 135 to 72.

irritability of the myocardium, extrasystoles often appear as the result of digitalis dosage and a bigeminal pulse, that is, an extrasystole coming shortly after each regular contraction is indicative of a toxic action from digitalis. Digitalis probably never decreases the size of a chronically enlarged heart sufficiently for it to be demonstrated by percussion. The important evidence of digitalis action on the heart is to be found in the amelioration of those symptoms caused by cardiac inefficiency, though change in the pulse rate often is a valuable criterion of digitalis action.

Digitalis produces a diuresis in cardiac patients with edema (Figs. 1, 3, 6, 7, 8). The exceptions are patients whose cardiac condition is so bad that digitalis produces practically no improvement in condition and patients with nephritis in addition to cardiac disease in whom renal function is too poor to allow of diuresis. It is very generally agreed that digitalis does not produce a diuresis unless cardiac edema is present. There is, however, considerable difference of opinion as to whether digitalis acts to produce a diuresis because it improves renal circulation as a part of the improvement in the general circulation, or because it has a direct diuretic action on the kidney. The better evidence, however, seems to indicate that diuresis is not the result of a direct diuretic action on the kidney. My own opinion is that digitalis acts indirectly as a diuretic but that this effect is so constant a result in the presence of cardiac edema that diuresis becomes a very important index of digitalis effect. So it is important to quantitate the daily urine output in relation to fluid intake as a measure of digitalis action, and to weigh patients at frequent intervals, decrease in weight being an important criterion of digitalis effect (Figs. 5, 6, 7, 8), even when diuresis is not marked (Fig. 9).

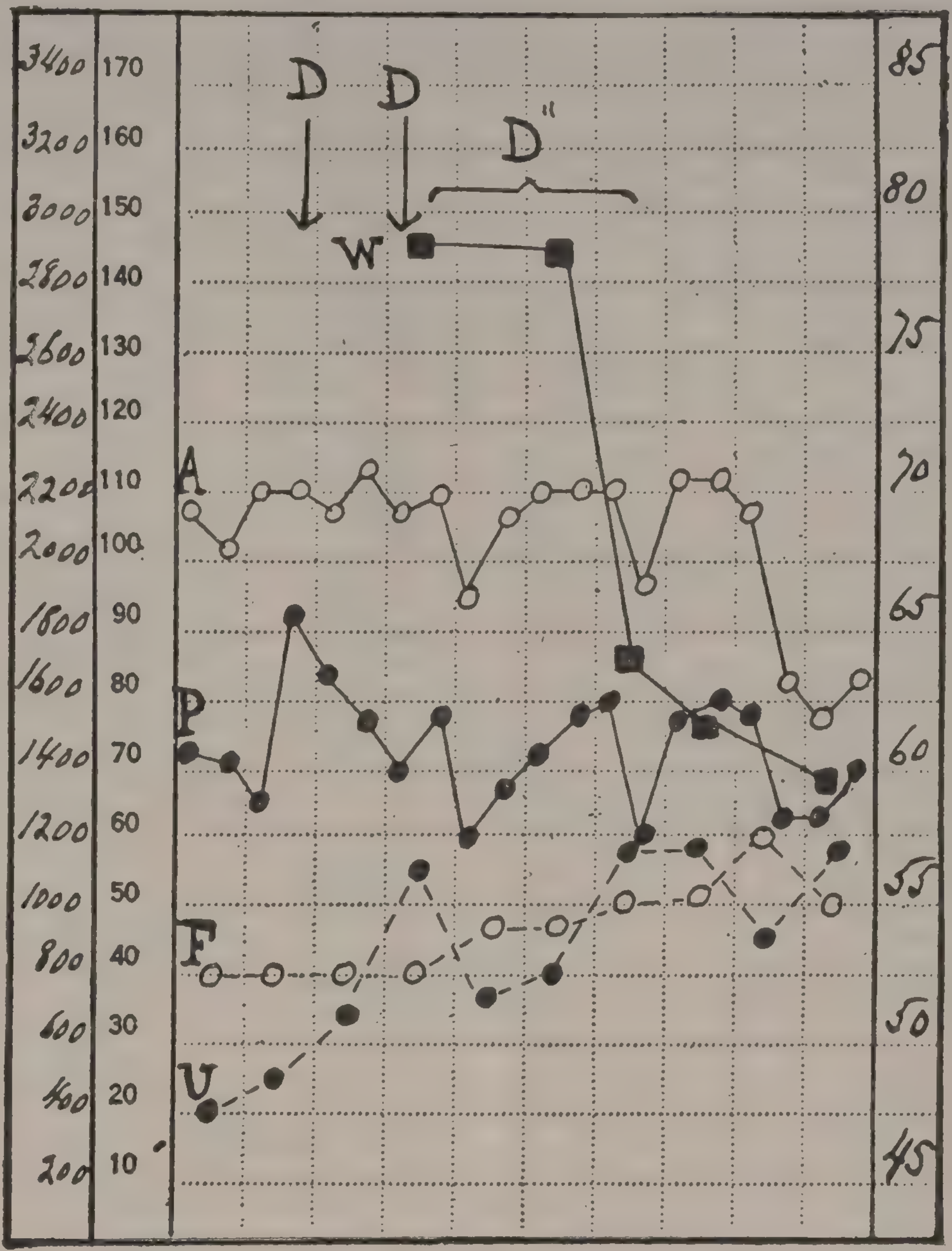


FIG. 5.—MALE, AGE 59, CHRONIC MYOCARDITIS, AURICULAR FIBRILLATION, ARTERIOSCLEROSIS, OLD RIGHT HEMIPLEGIA. The first column of figures on the left hand side of the chart indicates the amount of urine output and the fluid intake for each 24 hours expressed in c.c. The second column of figures on the left of the chart indicates the apex and radial pulse rates per minute. The column of figures on the right indicates the weight of the patient in kg. D = a single dose of 1.8 gm. of powdered digitalis leaves. D^1 = 0.2 gm. of powdered digitalis leaves. D^2 = 15 doses of 0.1 gm. each of powdered digitalis leaves every 6 hours, a total of 1.5 gm. Total $D + D^1 + D^2 = 3.5$ gm. of powdered digitalis leaves. A = heart rate counted with a stethoscope over the apex region. P = pulse rate counted at the wrist. F = fluid intake measured in c.c. U = urine measured in c.c. W = weight of the patient in kilograms. The effect of digitalis in this case was a delayed decrease in apex rate (110 to 78), with a moderate decrease in pulse deficit, a delayed but prolonged moderate diuresis, and a decrease in body weight of 19 kg., or 41.8 pounds. This required more than the usual amount of digitalis.

Digitalis, when given in sufficient dosage, causes nausea and often vomiting. Preceding the nausea usually there is loss of appetite, which, if carefully inquired after, can serve as a warning of impending nausea and, if digitalis is stopped, nausea may be prevented and yet excellent digitalis effects be obtained. To my mind nausea is a useful effect of digitalis; not that it is desirable to have our patients nauseated and vomiting, but because nausea and vomiting serve a very useful function in indicating sufficiency of digitalis and preventing serious toxic disturbances from giving too much. Nausea and vomiting are not the result of the local effect of digitalis preparations on the mucous membrane of the stomach, but a central effect through the vomiting center. Recent work seems to show that this is not a direct local effect on the vomiting center from digitalis in the circulation, but that digitalis bodies cause reflex nausea and vomiting through their direct action on the heart, the afferent impulses passing from the heart to the medulla mainly by way of the sympathetic nerves. At any rate, it seems quite clear that it is not the character of the digitalis preparation as a nauseating drug that is significant and it is extremely probable that digitalis preparations that will not produce nausea are deficient in digitalis activity of all sorts. Digitalis may, however, in some patients cause nausea as a result of taste associations, much as marshmallow cake may cause nausea to the patient who once has had the experience of having eaten too much of it and been made acutely ill with nausea and vomiting.

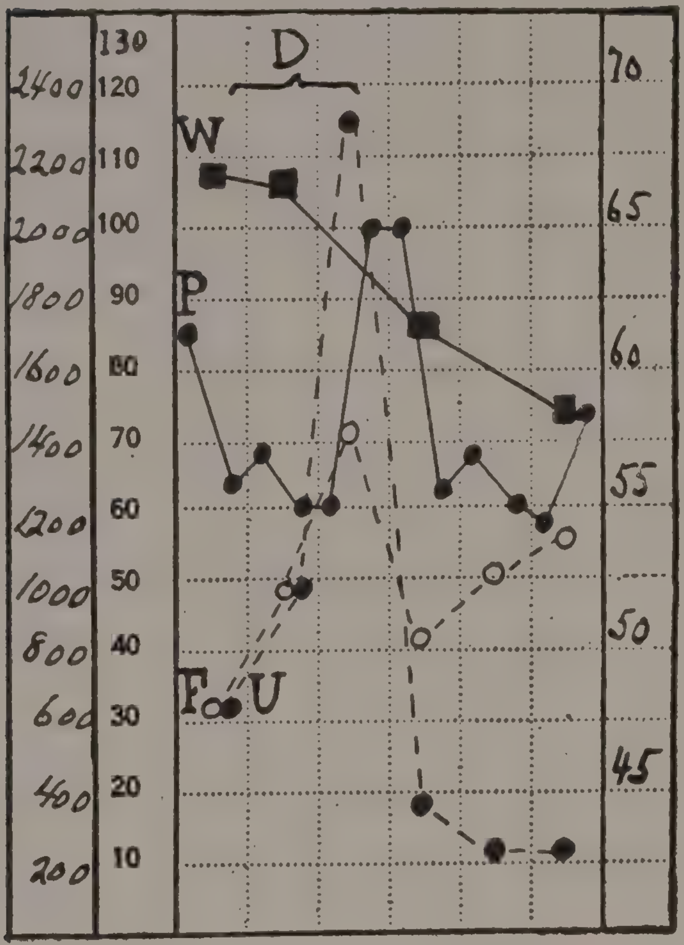


Fig. 6.—Male, Age 60, Chronic Myocarditis, Rhythm Regular. The first column of figures on the left hand side of the chart indicates the amount of urine output and the fluid intake for each 24 hours expressed in c.c. The second column of figures on the left of the chart indicates the apex and radial pulse rates per minute. The column of figures on the right indicates the weight of the patient in kilograms. D = 8 doses of 0.2 gm. each of powdered digitalis leaves every 6 hours, a total of 1.6 gm. P = pulse rate counted at the wrist. F = fluid intake measured in c.c. U = urine measured in c.c. W = weight of the patient in kg. The effect of digitalis in this case was a moderately slowed pulse rate (85 to 60), a diuresis with urine increase from 625 to 975 and 2,300 c.c., and a decrease in body weight of 7.8 kg., or 17.2 pounds.

As digitalis is given at present, it is rare for it to have any effect on the bowels; very occasionally diarrhea is produced.

Dosage.—In the words of Withering let digitalis "be continued until it either acts on the kidneys, the stomach, the pulse or the bowels." How are these ends best to be obtained? The answer is simple: Give digitalis in sufficiently large doses to produce some one of these effects, but stop short of rendering your patient uncomfortable from vomiting or from producing other toxic effects. Failure to obtain digitalis effects is by far a more serious error than to obtain a mild or even fairly severe toxic action. Damaging toxic digitalis effects in my experience, are extremely rare and can be easily avoided. Undue stress by so many medical writers on toxic effects has, I think, been a potent factor in causing the poor digitalis therapy that one so often encounters among medical men. The other important factor is regarding a drop as a minim and so actually giving patients much less tincture of digitalis than one thinks he is prescribing. Different digitalis tinctures will drop from 40 to 60 drops per 15 minims (1 c.c.); in other words vary from 2½ to 4 drops per minim. Liquid digitalis preparations should be prescribed to be taken by actual measure. Directing that so many drops are to be taken should be entirely discarded.

Sufficient digitalis in the sense of producing the desirable therapeutic effect can be attained in any one of three ways: the single large dose or body-weight method, the modified

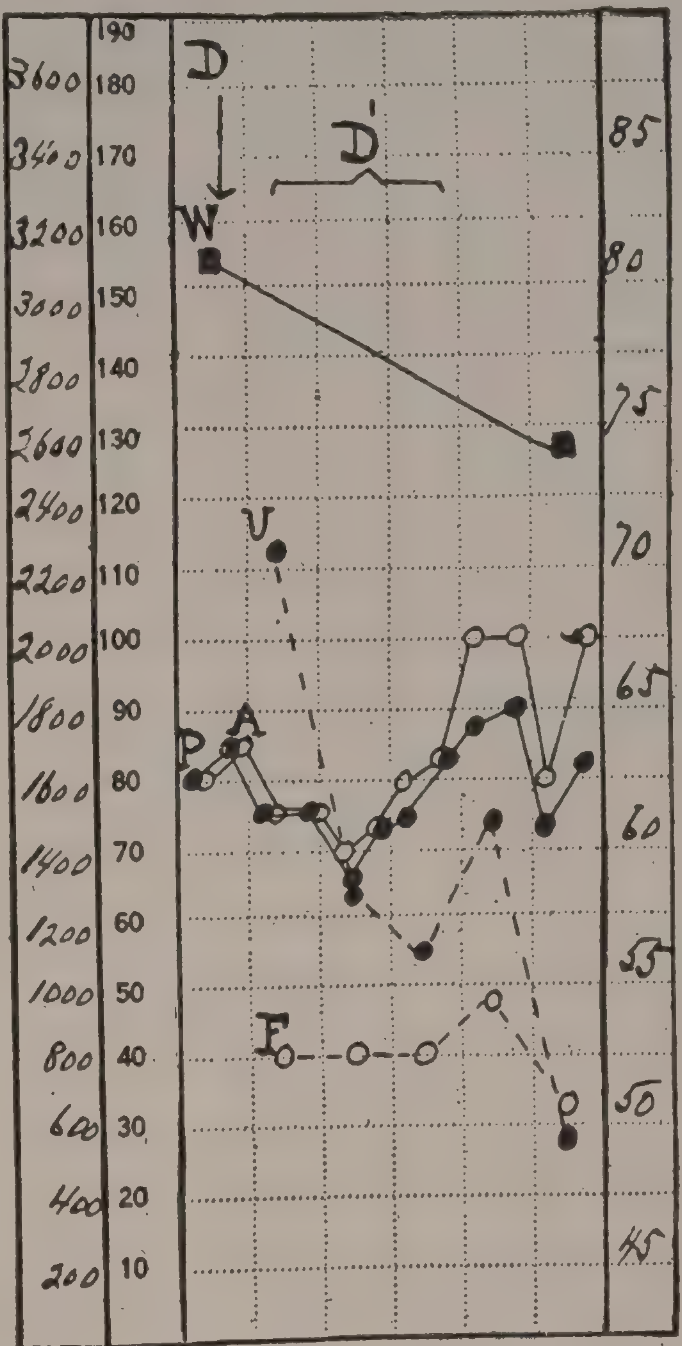


FIG. 7.—FEMALE, AGE 45, CHRONIC MYOCARDITIS, AURICULAR FIBRILLATION. The first column of figures on the left hand side of the chart indicates the amount of urine output and the fluid intake for each 24 hours expressed in c.c. The second column of figures on the left of the chart indicates the apex and radial pulse rates per minute. The column of figures on the right indicates the weight of the patient in kg. D = 0.5 gm. of powdered digitalis leaves given at 3.35 P. M. $D^1 = 9$ doses of 0.2 gm. each of powdered digitalis leaves given 4 times a day, a total of 1.8 gm. Total $D + D^1 = 2.3$ gm. of powdered digitalis leaves. A = heart rate counted with a stethoscope over the apex region. P = pulse rate counted at the wrist. F = fluid intake measured in c.c. U = urine measured in c.c. W = weight of the patient in kilograms. The effect of digitalis in this case was a diuresis with urine output of 2,250 c.c. and a decrease in body weight of 7 kg., or 15.4 pounds.

large dose method, the small divided dose method. The chief difference in the results from these several methods lies in the time needed to secure an effect. My preference is for the modified large dose method for the majority of patients, they being quite uncomfortable, the single large dose method for the very occasional patient who is extremely ill and has never had digitalis previously, and the divided dose method for such patients as are only moderately uncomfortable.

The Single Large Dose or Body-weight Method.—By this method the entire therapeutic dose is given at once (Fig. 8). The amount is calculated from the patient's weight and the activity of the drug according to the method of Eggleston. The latter is determined in cats by the method of Hatcher, the unit being the weight of dry drug in milligrams which is required to kill 1 kg. of cat when injected intravenously slowly and continuously. This is called a cat unit. The average total dose for man by mouth is 0.15 of one cat unit per pound of body weight. For calculating the actual dosage the following formulæ are used:

$$\frac{\text{C. U.} \times 0.15 \times \text{W.}}{1000} = \text{gm. powdered leaf.}$$

$$\frac{\text{C. U.} \times 0.15 \times \text{W.}}{100} = \text{c.c. tincture of digitalis.}$$

$$\frac{\text{C. U.} \times 0.15 \times \text{W.}}{10} = \text{c.c. infusion of digitalis.}$$

In these formulæ C. U. = number of milligrams in one cat unit, and W. = weight of patient in pounds.

Practically the body-weight method reduces itself to a guess at the patient's weight for the amount of digitalis given should be in ratio to the weight of the patient less the weight of the edema. The latter may be as great as 20 to 25 kg. (42 to 55 lb.) and this amount, as estimated, needs to be deducted from the actual weight. This being so it is hardly more accurate to weigh the patient in determining his weight than to guess at it from his height and apparent nutrition. As an average cat unit value for good digitalis is 100 mg., this gives a dose of 2.25 gm. of powdered digitalis leaves for a patient weighing 150 pounds. With this as a basis the proper dosage can be readily approximated for any patient, and to find the dose in cubic centimeters of tincture one merely has to multiply the above figure by 10 and in cubic centimeters of infusion by 100.

Caution needs to be observed to be sure that using this method one does not give a large dose of digitalis to one already having received digitalis and so partially digitalized. In practice this method should be reserved for the very occasional cardiac *in extremis* in whom a very prompt digitalis effect is necessary to save life. By giving such a single large

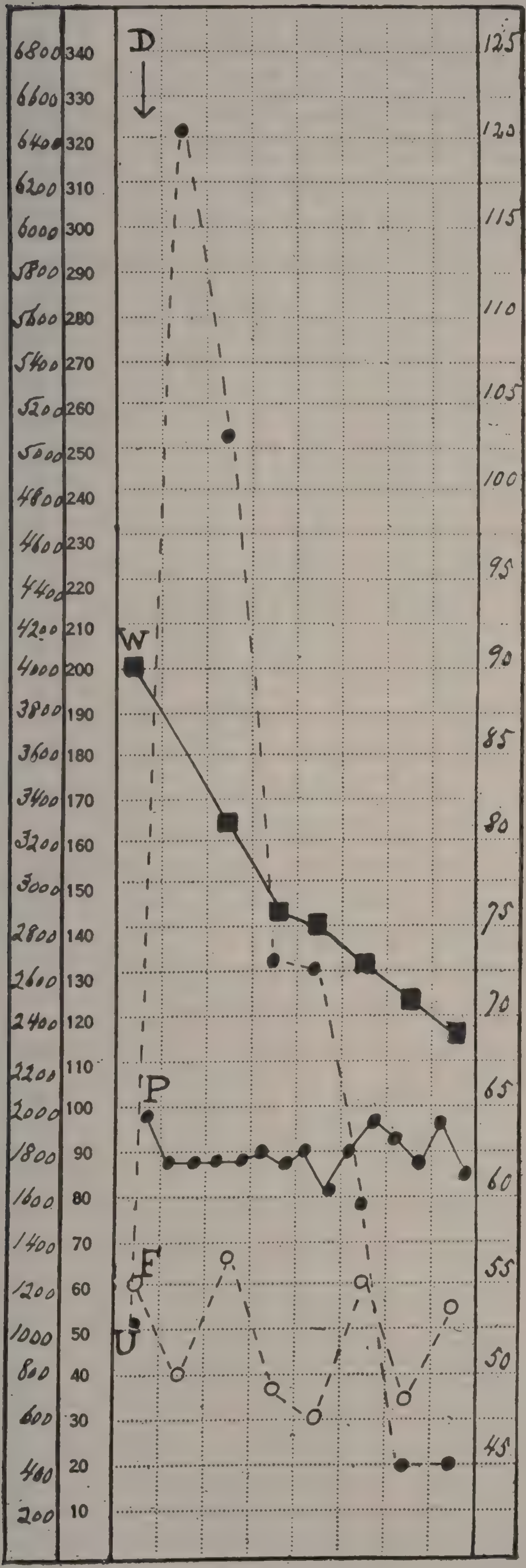


FIG. 8.—MALE, AGE 45, CHRONIC MYOCARDITIS, HYPERTENSION, RHYTHM REGULAR. The first column of figures on the left hand side of the chart indicates the amount of urine output and the fluid intake for each 24 hours expressed in c.c. The second column of figures on the left of the chart indicates the apex and radial pulse rates per minute. The column of figures on the right indicates the weight of the patient in kilograms. D = a single dose of 2.3 gm. of powdered digitalis leaves given at 10.30 A. M. P = pulse rate counted at the wrist. F = fluid intake measured in c.c. U = urine measured in c.c. W = weight of the patient in kg. The effect of digitalis in this case was to produce a very marked diuresis with increase of urine from 1,000 to 6,425, 5,050, 2,625 and 2,600 c.c., and a decrease in body weight of 21.4 kg., or 47 pounds.

dose considerable digitalization is attained in six hours or even less time and this may be life-saving in a rare patient.

The Modified Large Dose Method.—The average amount of powdered digitalis leaves required to digitalize a small patient, if the drug is given in a short period of time by the modified large dose method, is 2 gm.; for a medium-sized patient is 2.25 gm. and for a large patient is 2.5 gm. By the modified large dose method one-fourth of this total dose is given and the same dose repeated in four hours. The remainder of the amount needed is given at the rate of 0.1 to 0.2 gm. every four hours (Fig. 2). For a small patient this would be 0.5 gm. + 0.5 gm. four hours later + five doses of 0.2 gm. each every four hours. At the end of twenty-four hours the patient would have received his last dose of this series. For some patients this amount proves insufficient and for these further digitalis should be given at the rate of 0.1 gm. of powdered leaves three times a day until there is beginning nausea or other evidence of digitalis effect.

The Small Divided Dose Method.—By this method either 0.2 gm. or 0.1 gm. of powdered digitalis leaves is given three times a day or, more often, either every four hours or six hours until the signs of digitalis action appear (Figs. 3, 4 and 6).

Obviously in the last two methods, it is not necessary to follow any one fixed plan of dosage but considerable modification can be made (Figs. 1, 5, 7 and 9) in accordance with the need of speed in digitalization and previous digitalis therapy. Moreover, the actual amount per dose will need to be modified in accordance with the actual potency of the preparation used. The more divided the dose, the longer the time required for complete digitalization. However, slighter evidences of digitalis action are more easily detected in the small divided dose method, and it is easier then to determine just how to continue digitalis action and far easier to anticipate and so prevent uncomfortable digitalis effects, especially nausea and vomiting. The more one intelligently particularizes digitalis therapy for the individual case, the more satisfactory the result. Were I asked to sum up briefly the comparative effects of these methods, I would say the larger the amount of digitalis given in a short time the more dramatic the effect, but the small divided dose method means fewer nauseated patients, and except for the extremely decompensated ones, better satisfied patients.

In all of the above it is to be remembered that the smallest dose recommended, 0.1 gm. (1½ gr.) of powdered leaves, equal to 1 c.c. or 15 minims, or from 40 to 60 drops of tincture, is really a large dose as digitalis therapy is generally in vogue.

Digitalis Failures.—Every cardiac patient, sooner or later, will reach the stage of no response to digitalis because the heart muscle is no longer able, by reason of its disease, to increase systolic output. The remarkable thing is that this stage is so long delayed in most patients and that the diseased heart will continue to respond to digitalis. The important thing for the patient is to be sure that the stage of no heart response has been reached instead of insufficient digitalis having been given and absorbed. The first step toward this certainty lies in knowing that you are giving potent digitalis in sufficient dosage; the next step in knowing that it is being absorbed. If one is using the same digitalis in several cardiac cases, and is obtaining good digitalis effects in all but one, he has the complete surety of potent digitalis and sufficient dosage if he continues to use that digitalis preparation. The next best index of digitalis potency lies in its having been recently tested on animals by a reliable laboratory. Under these circumstances, lack of absorption or inability of the heart muscle to respond are the only explanations of failure to obtain digitalis effects. A further check can be made by insuring absorption through giving some of the digitalis group intravenously (this method of medication is described later). This having been done without obtaining a digitalis effect, it is fair to say that the heart has lost its ability for a digi-

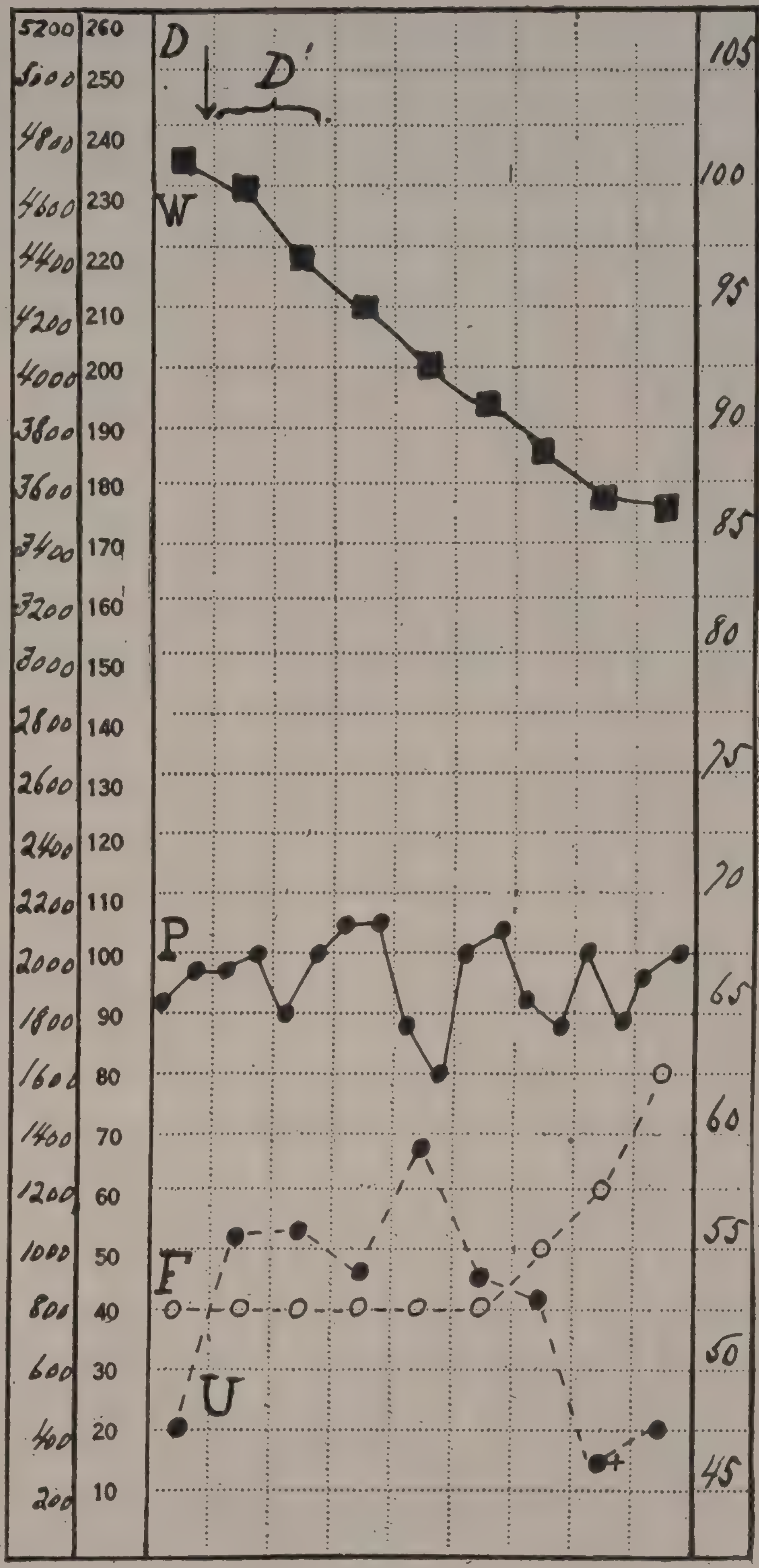


FIG. 9.—FEMALE, AGE 43, CHRONIC MYOCARDITIS, HYPERTENSION, RHYTHM REGULAR. See Fig. 8 for explanation of figures in Chart. D = a single dose of 1.2 gm. of powdered digitalis leaves given at 9.30 P. M. $D^1 = 5$ doses of 0.2 gm. each of powdered digitalis leaves every 6 hours, started at 3.30 A. M., a total of 1 gm. Total $D + D^1 = 2.2$ gm. of powdered digitalis leaves. P = pulse rate counted at the wrist. F = fluid intake measured in c.c. U = urine measured in c.c. W = weight of the patient in kg. The effect of digitalis in this case was a slight prolonged diuresis and a decrease in body weight of 15 kg., or 33 pounds.

talis response. Sometimes intestinal edema prevents absorption and brisk saline catharsis will be followed by good digitalis absorption when the digitalis is given after the catharsis. As I see cardiac cases, poor absorption plays a surprisingly minor rôle in explaining poor digitalis effects.

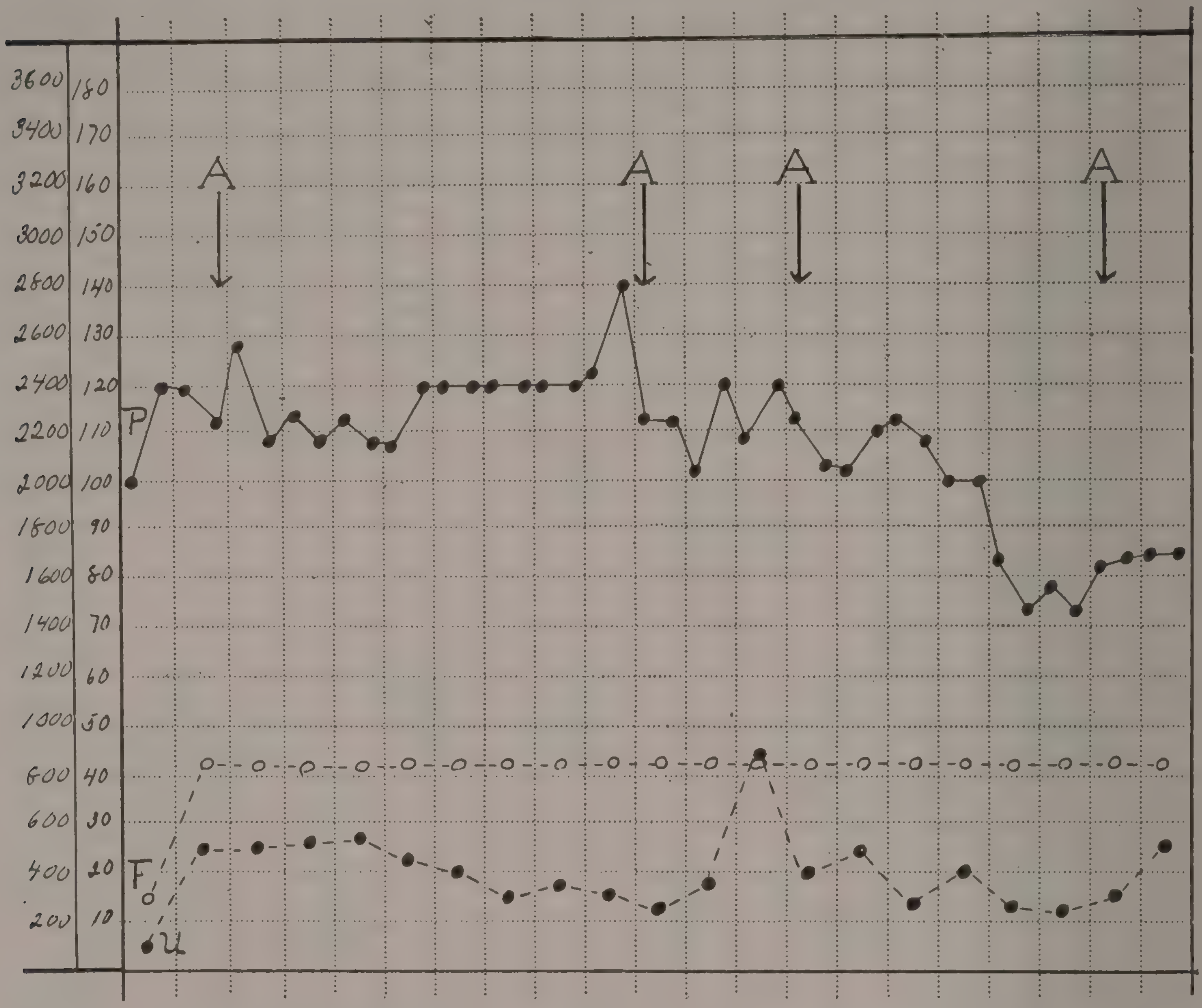


FIG. 10.—MALE, AGE 56, CHRONIC MYOCARDITIS, RHYTHM REGULAR. The first column of figures on the left hand side of the chart indicates the amount of urine output and the fluid intake each twenty-four hours expressed in c.c. The second column of figures on the left hand side of the chart indicates the pulse rate per minute. A = a single intravenous dose of 1 mg. of amorphous strophanthin. P = pulse rate counted at the wrist. F = fluid intake measured in c.c. U = urine measured in c.c. The effect of intravenous strophanthin is shown in a decrease in the heart rate from an average of 120 to an average of 80 and a moderate diuresis.

Vomiting, obviously, will hinder digitalis absorption. How to treat the vomiting patient will be described later.

Intravenous and Intramuscular Digitalis Therapy.—The intravenous route of dosage is preferable to the intramuscular because of lack of local irritation and quicker absorption. Subcutaneous use of digitalis preparations should not be attempted on account of the frequent uncomfortable local reactions. For intravenous use amorphous strophanthin (Fig. 10) or crystalline ouabain are best. Of the former 0.5 mg. is the average dose and 1 mg. should never be exceeded. In fact 1 mg. is usually a sufficient amount for twenty-four hours whether given at once or in divided doses; sometimes as much as 3 mg. may be used in twenty-four hours. Ouabain is more toxic and its dose should be from two-thirds to one-half that of amorphous strophanthin. Both are too irritating for satisfactory intramuscular use. In place of these, liquid digipuratum (1 c.c. is the average dose) or other forms of digitalis, dispensed for intravenous use, may be employed. It is to be remembered that in emergency, when none of these preparations are available, pharmacopeial tincture of digitalis can be used intravenously in 1 to 2 c.c. dose (Fig. 2). In all intravenous digitalis therapy it is well to give the drugs well diluted. This is especially true of strophanthin and ouabain. Eggleston advises at least 1:1,000 dilution of the former and 1:4,000 of the latter in normal salt solution. Digitalis preparations of any kind should not be given intravenously to patients who recently have been taking digitalis by mouth; great caution in this respect is imperative when using strophanthin or ouabain.

In patients in whom the veins cannot be entered by the injection needle liquid digipuratum or similar preparations may be given intramuscularly (Fig. 4). As for intravenous use, tincture of digitalis may be substituted when other preparations are not available, even though local irritation will result.

Digitalis in the Vomiting Cardiac.—The cardiac patient may vomit as a result of needing digitalis or by reason of having had too much digitalis. In other words, cardiac decompensation may be causing vomiting or digitalis may be producing vomiting as part of its pharmacologic activity. Obviously, it is important to distinguish between these. If vomiting results from digitalis activity discontinue the drug and in twenty-four hours the vomiting will largely cease. It is not likely that, under these circumstances, a change to another of the digitalis group will influence vomiting unless the new drug has less of a digitalis action or is not absorbed. The former practice under these circumstances of substituting tincture of strophanthus for tincture of digitalis is now easily explained; vomiting ceased because the strophanthus was very poorly absorbed; some other similarly inert drug would have produced the same result. In the same way giving digitalis intramuscularly or intravenously is not a logical procedure to obviate vomiting caused by digitalis activity, since the vomiting is a reflex mechanism through the vomiting center and caused equally, whatever the route of dosage, when a given amount of digitalis gets into the system. In the same sense various special digitalis preparations claimed to obviate digitalis vomiting are illogical.

In this connection it is important to distinguish between vomiting due to digitalis activity and vomiting from the taste, or even odor, or in rare cases the merest thought of digitalis. This sort of vomiting is an idiosyncrasy usually acquired as a result of having undergone nausea and vomiting as a result of a previous course of digitalis treatment. With such patients digitalis may be given successfully either intravenously or intramuscularly. Not infrequently merely placing the powdered digitalis leaves in capsules, being careful to remove all digitalis from the outside of the capsule, is all that is needed to overcome the nausea and vomiting. To use some pleasantly flavored fluid to wash the capsule down is helpful. Often, however, the capsule needs to be prepared so it will not dissolve in the stomach. This may be accomplished by coating it with salol. Gelatin capsules, too, may be rendered insoluble in gastric juice by treatment with formaldehyd, the so-called glutoid capsules of Sahli.

Very rarely there is a patient to whom digitalis can in no way be given by mouth owing to the patient's very great susceptibility to it in the sense of causing vomiting. What I want to emphasize, however, is that these are extremely rare patients. In my experience I have found that almost always digitalis can be given to patients so as to obtain a therapeutic effect so long as the heart remains responsive to digitalis.

When vomiting is a symptom of cardiac insufficiency, usually digitalis can be given merely by timing the doses so that they are given in periods of gastric quiescence rather than by the clock, and given this way it will be retained. Failing this, digitalis should be given intravenously or intramuscularly until its action stops the vomiting and then by mouth to complete the amount needed for the desired digitalization. Caution is necessary to prevent toxic effects from giving the drug intravenously or subcutaneously after digitalis has been given by mouth.

Evidence of Too Much Digitalis.—The most frequent sign of too much digitalis is nausea and vomiting and when occurring they indicate cessation of the drug. In addition there are certain disturbances in rhythm which indicate that too much digitalis has been given. A not infrequent one of these and also one easily recognized by the palpating finger is a bigeminal pulse. This is due to regularly recurring extrasystoles, one coming shortly after each regular contraction and followed by a slightly longer pause than the one preceding it. As the extrasystole usually causes a smaller systolic output, the pulse wave is smaller giving a pseudo pulsus alternans. This arhythmia is seen perhaps oftenest in auricular fibrillation after digitalis has markedly slowed the pulse. It must be remembered, however, that the same arhythmia may occur without digitalis, being due to myocardial lesion. In the latter event, digitalis should be given whereas, when it develops after digitalis therapy, digitalis should be stopped.

Digitalis increases muscle irritability and is therefore sometimes followed by an increased number of irregularly occurring extrasystoles. As extrasystoles occur under so many conditions, it is difficult to interpret them. Unless they produce a bigeminal pulse, for the most part they should be ignored as far as digitalis therapy is concerned, except when there are other evidences of digitalis intoxication.

Digitalis may cause partial or complete heart-block. As a rule, this does not happen until late in digitalis therapy unless there is some lesion of the conduction system. Other rarer forms of arhythmia may result from digitalis but these are not of sufficient practical importance to justify description.

It is to be remembered that when digitalis is being given in any reasonable dosage and the patient is being watched carefully, there is little chance of any harm coming to the patient because evidences of too much digitalis appear in definite enough form to indicate this and permit discontinuance of the digitalis before any real harm has been done.

Other Drugs with Digitalislike Action.—Other drugs with a digitalislike action are strophanthus, convallaria, squill and apocynum. These have largely and rightly fallen into disrepute for treating cardiac cases because they are very poorly absorbed or give unsatisfactory results. I find no occasion for their use by mouth. Amorphous strophanthin and ouabain are valuable in the occasional case for intravenous use when digitalis is not absorbed or cannot be retained on the stomach. With this exception these other drugs should be entirely discarded.

Other Drugs Acting on the Circulation.—*Caffein* is of little use in decompensation of chronic cardiac disease except that infrequently it is useful as a diuretic. In large doses it may improve respiration, converting a Cheyne-Stokes type into a regular respiration, but its other effects are, on the whole, too disturbing to make it of any real service in the therapy of the chronic cardiac.

Strychnia is of little or no use. *Camphor* is almost inert except for a transitory reflex stimulation of the heart when given subcutaneously in oil, preferably mixed with ether. Neither of these drugs has any real place in the treatment of cardiac disease.

Nitroglycerin is often combined with digitalis therapy in treating the patient with hypertension, but I have never seen any advantage from the nitroglycerin and the same applies to the nitrites. For the anginal attacks of the chronic cardiac, however, nitroglycerin may be of much value.

Quinidin is described under special treatment of auricular fibrillation.

Drugs for Restlessness and Insomnia.—The use of morphin to give a good night's rest in the first days of treatment of the badly decompensated cardiac has been described previously. Its use in this way constitutes a very important part of the therapeutics of heart disease.

Continued nervousness, restlessness and insomnia not controlled by the other measures of treatment often require some drug. Potassium bromid in 1 gm., or larger doses, three times a day frequently is useful. Sir James Mackenzie has a strong preference for ammonium bromid instead. Chloral, 0.3 to 0.6 gm. and repeated once or twice at two-hour intervals, barbital (veronal) or its sodium salt, 0.3 gm. to 1.0 gm., sulphonmethan (sulphonal) 1.0 gm., all may be tried. Morphin may be needed in oft-repeated doses in the patient beyond much response to digitalis. Yet it is to be remembered that the patient is better off without any of these drugs, if they can be omitted, and that sometimes they actually excite. It is well to try a night without any drugs every few days, giving the patient a placebo in appearance and route of dosage similar to the discarded drug, if the patient is susceptible to the psychology of a supposed dependence on the drug. It has always seemed to me that the most skillful physicians manage their cardiacs with the least use of these drugs and that much use of them marks failure in handling the circulatory situation. The really skillful physician is only forced to admit failure for those patients in whose hearts disease has progressed beyond response to cardiac drugs, a stage that, alas, sooner or later all cardiacs will reach unless they die earlier from some intercurrent disease.

Catharsis.—Except for such mild cathartics, really laxatives, as are needed to insure a daily bowel movement of approximately normal character, most cardiacs do not require catharsis. Active watery catharsis as a means of removing edema should be regarded as a measure to be avoided because it interferes with the measures described for the patient's rest, because it is a procedure unpleasant to both patient and attendants, because it is debilitating and because it is not needed for most patients. Frequent defecation is both more uncomfortable and less esthetic and so less desirable than frequent urination as a channel for removal of fluid from a cardiac. There is a very small group of cardiac patients, already described, in whom digitalis absorption is poor and in whom following active catharsis it becomes good. These should have magnesium sulphate in a 30 to 60 gm. dose dissolved in a moderate amount of water in the early morning for one to two days. There is a somewhat larger group in whom edema, particularly ascites, seems to hinder mechanically its removal by the kidney but in whom catharsis is effective. These should receive similar management with magnesium sulphate but with failure after a few days, resort needs to be had to mechanical removal of fluid as described further on. There is a still larger group of cardiacs with poor renal function by reason of a concomitant nephritis in whom diuresis cannot be produced. These patients should have daily catharsis with magnesium sulphate as long as they will stand it and for variety may be changed to compound jalap powder in 2 gm. doses. However, after deducting all of these, there remains the very large majority of cardiac patients who need no catharsis because with rest, digitalis and limited fluid intake they have diuresis sufficient to remove all edema and that is the only reason for active watery catharsis. To these give, if needed, extract of cascara or liquid petroleum or similar laxatives to produce one daily stool.

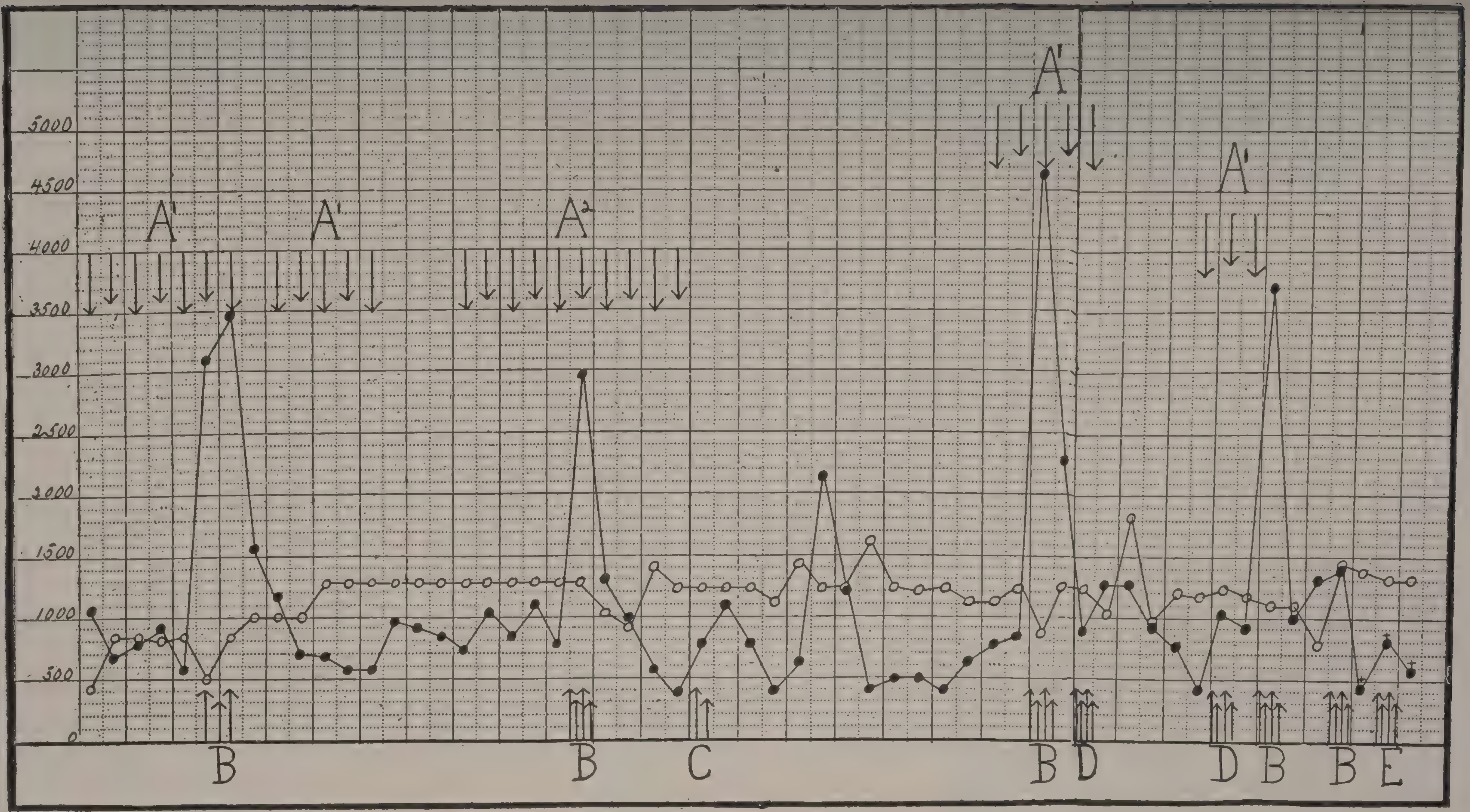


FIG. 11.—PATIENT WITH AORTIC STENOSIS AND INSUFFICIENCY. The solid black dots indicate the 24-hour amount of urine in c.c. The black circles indicate the 24-hour amount of fluid intake in c.c. Each arrow under A^1 indicates a day on which the patient received three doses of 10 c.c. each of an infusion of digitalis. Each arrow under A^2 indicates a day on which the patient received two doses of 10 c.c. each of an infusion of digitalis. Each arrow over B indicates one dose of 0.5 gm. of theophyllin (theocin). Each arrow over C indicates one dose of 0.2 gm. of caffein sodiobenzoate. Each arrow over D indicates one dose of 0.3 gm. of theobromin sodiosalicylate. Each arrow over E indicates one dose of 0.5 gm. of theocin sodioacetate. Note that marked diuresis follows theocin; that there is a very slight diuresis after caffein sodiobenzoate; that there is no diuresis after theobromin sodiosalicylate, even though theocin given the next day causes a marked diuresis.

Diuresis.—For the great majority of cardiacs with edema, digitalis, given as already described, is a wonderful diuretic (Figs. 1, 3, 6 and 8) and all that is needed. Occasionally it needs to be helped. Theophyllin is the most active of the diuretics for such a patient (Fig. 11). It should be given in two to three doses of 0.2 to 0.3 gm. each at four hour intervals. Usually it need not be repeated but, if repetition is needed, this dosage should be given on alternate days. Occasionally doses as large as 0.5 to 0.7 gm. may be effective when smaller doses fail. Theophyllin is in the pharmacopeia but not in the pharmacy, so theocin, which is the trade name for theophyllin, will have to be used. Theobromin sodiosalicylate is next in activity. It should be given similarly but a larger dose may be used, 0.3 to 0.5 gm.; occasionally 1.0 gm. Both theocin (theophyllin) and theobromin may be given intravenously (Fig. 12). Caffein is less efficient for diuresis as a rule. Its dosage is 0.2 to 0.3 gm. However, in some patients one of these diuretics will be effective when another has not been, and so they may be tried in succession. Unfortunately, usually when they do not work, we are dealing with a nephritic condition along with the cardiac and

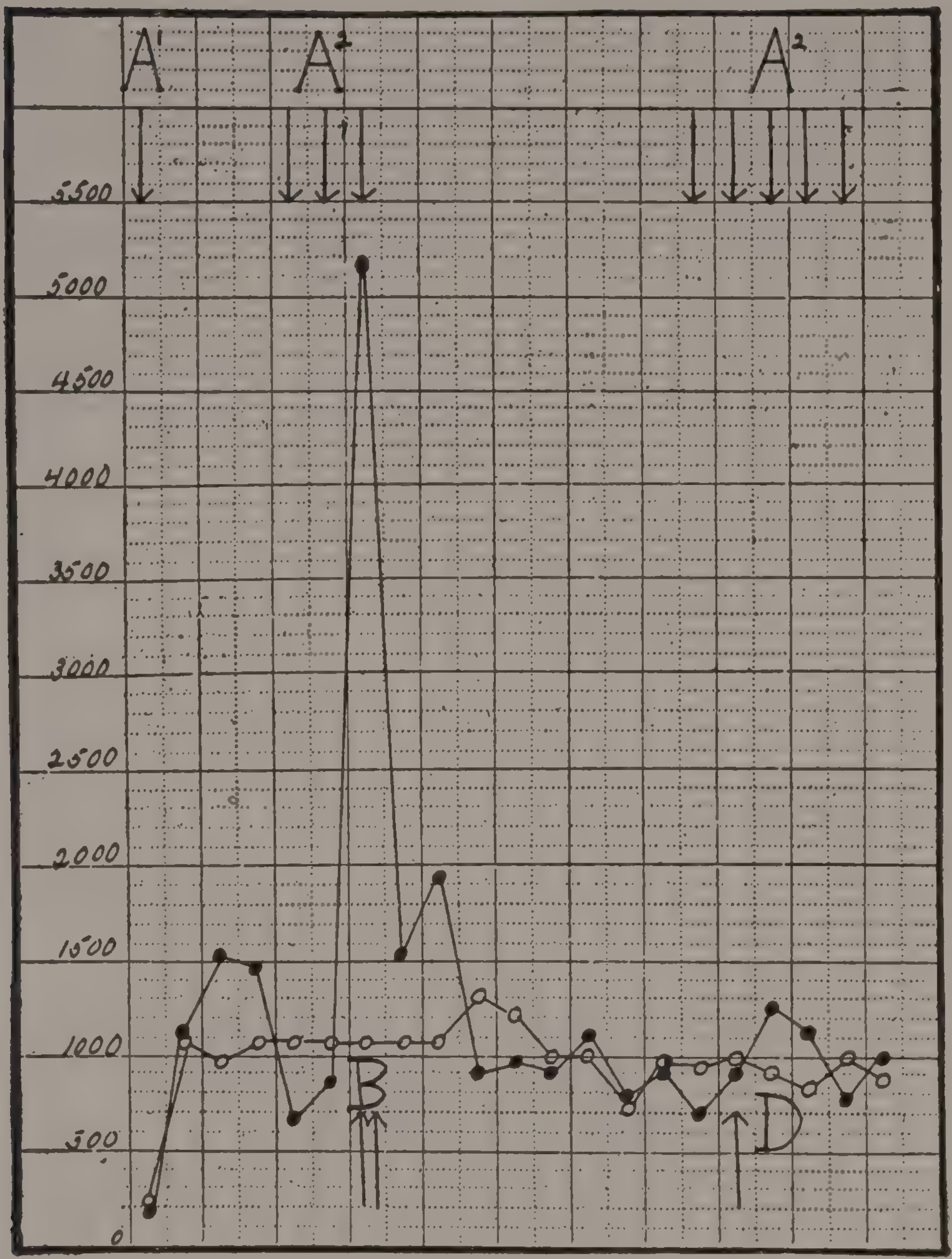


FIG. 12.—SAME PATIENT AS IN FIGURE 11. The solid black dots indicate the 24-hour amount of urine in c.c. The block circles indicate the 24-hour fluid intake in c.c. The arrow under A¹ indicates a subcutaneous dose of 1 c.c. of digipuratum. The arrows under A² indicate a day on which the patient received 10 c.c. of infusion of digitalis three times. The arrows at B indicate two doses of 0.5 gm. each of theocin given intravenously. The arrow at D indicates a dose of 1 gm. of theobromin sodiosalicylate given intravenously. Note that diuresis follows theocin and not theobromin, the same difference as noted when they were given by mouth as shown in Figure 11.

nothing will produce a diuresis; or with so marked an ascites that its pressure prevents an adequate circulation to the kidneys to allow of diuresis. In the latter, paracentesis is effective, at least temporarily, and sometimes after paracentesis a diuretic will work when previously it was ineffective.

Mechanical Removal of Fluid.—*Hydrothorax,* if extensive, even though of cardiac origin, is very little influenced by digitalis, diuretics or catharsis. It should be removed mechanically by the usual procedure of trochar and aspiration bottle and this should be done promptly. If the physical signs show dulness in the back to a point halfway between the spine and angle of the scapula I would tap as one of the opening moves of the patient's treatment. Recurring effusions should be removed as soon as they seem to be producing an added factor in the patient's discomfort.

Hydropericardium as part of cardiac decompensation, apart from pericarditis, is of no practical significance and requires no special management.

Ascites usually disappears with digitalis or with digitalis plus diuretics or cathartics. When it does not or when it causes evident respiratory or circulatory embarrassment, it should be withdrawn by trocar in the usual way. Recurrence to the point of producing a tensely distended abdomen or respiratory or circulatory distress is a sign for repetition of withdrawal.

Subcutaneous edema may be extensive enough to cause much discomfort by distending the skin and not infrequently by leading to secondary inflammatory changes. Edema of the genitalia is particularly apt to cause discomfort and may interfere with micturition. If not relieved by the various means already described, edema can be drained by means of Southey's tubes inserted deep in the edematous subcutaneous tissues of the lower legs. If Southey's tubes are not available, any form of trocar of moderate caliber may be substituted. The skin should be cleaned at the site of insertion with soap and water and alcohol; then tincture of iodin should be applied. The tubes are to be inserted obliquely so as to be as nearly as possible in the line of the leg. The tubes should be connected with rubber tubing leading into a bottle attached to the side of the bed to collect the fluid. By these means one or more liters of fluid will drain away over night. One or two tubes should be inserted in each leg. Another method is to make one or two generous incisions through the skin, cleaned as for the Southey tubes, deep into the edematous subcutaneous tissues of each lower leg. When the leg is tense with edema there is very little bleeding. Infection, as a rule, does not occur. Fluid drains freely from such incisions, so that the problem of absorbing the fluid is a real one. It is necessary to protect the bed with a rubber or linoleum sheet or the patient may sit in a chair with his feet in a foot tub. It is really very surprising how much fluid will drain through such punctures or incisions in the lower leg and how far it will come. Not only will the edema disappear from the upper legs but also from the scrotum and penis and from the trunk as high as the mid-thorax. I feel very sure that such mechanical drainage of subcutaneous edema could be carried out much oftener than is commonly done with benefit to the patient. At times this is the only method that will be effective in removing edema, but once removed other methods prevent its recurrence until another period of decompensation comes.

Bleeding.—For marked cyanosis and its attendant dyspnea bleeding often is of very great value, bringing prompt relief; from 250 to 700 c.c. of blood should be withdrawn. The best method is to insert a hollow needle into one of the larger veins of the arm, using a tourniquet if necessary to make the vein more prominent. Gentle suction by using a bottle with a two-way cork will facilitate the flow of blood. One should remember the direction of flow of venous blood and if there is difficulty in obtaining blood with the needle inserted in the vein toward the body, insert it toward the hand. Cutting down on and incising the vein is efficacious and can be done when, for one reason or another, the needle puncture method cannot be carried out. Old-fashioned wet cupping also is effective and should be used when, as sometimes occurs, no satisfactory flow of venous blood can be obtained from the arm veins by either puncture or incision method. I am strongly of the opinion that bleeding is far too little used at present in the management of the severely decompensated cardiac.

Diet.—For the edematous cardiac during the first four days of treatment the so-called Karell diet is admirable in its extreme simplicity and effectiveness. This consists of a total of 800 c.c. of milk per twenty-four hours (four ordinary glasses of milk). With this diet and rest in bed without any medication one often sees a marked diuresis and other changes in symptoms and signs. The Karell diet obviously is a starvation diet and should be maintained but a few days.

After the first few days the patient should be on a simple, bland, easily taken diet with total fluids restricted to 1,000 to 1,500 c.c. per twenty-four hours. The total amount should not be great. Frequent feedings are desirable. There are no special limitations needed as to the diet except digestibility. The dyspneic patient cannot use dry foods very well and such as require much chewing are undesirable. Excess of carbohydrate often causes too much intestinal gas and so carbohydrates must be diminished. It is well to remember that not infrequently solid food will be retained by a patient who persistently has vomited liquids and soft solids. Salty food, though generally undesirable, may be the only sort of food that will be retained. Frequently I have stopped nausea and vomiting in my patients, greatly to the surprise of my house-officers and students, by allowing the patient to have ham, generously salted cold chicken or lamb, salt fish, or smoked salmon, some of these seemingly very indigestible foods but, when craved by the patient, not causing vomiting and so far as could be told, being digested perfectly. For the nauseated patient iced, dry ginger ale or equal parts of ginger ale and milk often are very satisfying and the latter has considerable nutritive value. The former is perhaps the best substitute in the United States for the iced champagne, now interdicted by our zealous lawmakers, which in the past often worked wonders for the nauseated, vomiting patient.

Continuation Treatment

Having carried out the methods described under Treatment During the Stage of Heart Failure or Decompensation, and put the patient in condition to be practically free of symptoms while in bed, the next important consideration is to plan the management of the patient so as to obtain the maximum of efficiency with the minimum of cardiac damage. For this it is necessary to describe getting the patient out of bed, continued digitalis, permissible activity and exercise, etc., all of which can be expressed under the term "continuation treatment."

Getting the Patient out of Bed.—When should the cardiac case being treated for decompensation be allowed to get out of bed and how should the time allowed up be regulated? As a general rule, the cardiac patient should remain in bed until all signs of subcutaneous edema, ascites or pulmonary edema (fine râles at the base of the lungs) have disappeared and until most, if not all, the symptoms of cardiac disease have markedly decreased or disappeared. It is to be remembered that for the patient in bed subcutaneous edema lurks longest over the sacrum. Even if these signs and symptoms disappear promptly, it is a safe rule to keep any cardiac case, sick enough to be put to bed by you, in bed for at least one month and to increase this time in ratio to the severity of symptoms and signs.

The exception to the rule in bed until signs and symptoms have largely disappeared is that, if after a prolonged period of treatment in bed, no further improvement is noted in signs and symptoms, one should recognize the inability to restore satisfactory cardiac function and allow the patient to get out of bed for short periods of time. This will allow one to see whether or not the patient's general condition is not better for the change and exertion incident to getting up, remembering that one is dealing with a chronic, strictly speaking incurable disease with which the patient must be managed so as to have the greatest possible comfort and happiness, and that sometimes getting up furnishes an exercise that actually improves circulation. Optimism with the cardiac is very important, optimism based on the knowledge that most cardiac conditions can be greatly improved. Some laxity in restrictions, which are really needed, at times is required to maintain the patient's morale. In all of this, good clinical judgment is a great asset, for no definite indications can be given that will fit each and every patient. In this connection it is to be remembered that the size of the heart, the type of the murmur, or the arhythmia are no indications for denying or allowing increased activity. The important criterion is how well is the heart doing its work.

Getting out of bed always should be a gradual process. The first day have the patient helped to a chair near his bed to remain for fifteen minutes, then back to bed. If an hour later his pulse is back to its level before getting up and the patient has had no uncomfortable symptoms while up or afterwards, increase the time next day by fifteen minutes. On the third day let him up for an hour, on the fourth for an hour and a half, and on the fifth day for two hours. Then reduce the time but have the patient up twice a day beginning with thirty minutes for each time and gradually lengthening the time. Undue fatigue, persisting tachycardia, increasing edema and dyspnea are the best indications of when the period out of bed has been too long.

Walking about the room gradually should be added, at first with some one to help the patient. With the indications just stated one can be safely guided to allowing to the cardiac patient more and more freedom. Patients usually welcome going to the toilet as one of their early activities.

Continued Digitalis.—Many cardiac patients do far better when they take daily small doses of digitalis over long periods of time. The majority of patients will tolerate 0.1 gm. of powdered digitalis leaves every twenty-four hours over long periods of time, not a few will tolerate 0.2 gm., depending of course on the rate of elimination. This may be given in one dose each day or it may be divided into two doses. I rather prefer a morning and a night dose. The intelligent patient after some experience will learn to appreciate the early evidences of both too little and too much digitalis and can guide his dosage accordingly. This applies particularly to the patients constantly just on the border line of decompensation.

It seems to be a good plan to give such daily amounts of digitalis to those cardiacs who have been bed cases during the period of getting out of bed and trying varying activities. Later it may be omitted and careful observation will determine whether they do better with the drug or not. In some patients it seems desirable to give some digitalis daily for an indefinite time, increasing the amount if symptoms progress, but never discontinuing it. After digitalization of the bed patient I advise 0.05 gm. of powdered digitalis leaves night and morning until the patient is up and about all day. This may not prove enough but it is extremely rare for it to cause nausea or any other unpleasant digitalis effect. This small dosage does seem to continue the digitalis effect, once it has been obtained by a larger dosage, and for some patients it very definitely increases the range of their activity and so should be continued in use. I have never seen any disadvantage from such a prolonged use of digitalis as is described here.

Permissible Activity and Exercise.—What the cardiac patient, once bedridden, can do is a very varying thing and depends on many factors so complex that, without any very satisfactory tests of cardiac function, we must needs depend on experimentation for each patient, remembering that both too little and too much activity are harmful to heart muscle. During convalescence the patient must be tried at various activities. His future work must be proportioned to his cardiac ability. His home conditions as to stair-climbing, etc., as well as similar conditions at his place of work, enter into the problem. Mode of transportation from place to place is important. All of this essentially is a problem for the family physician, not for the consultant, for the latter is very apt to see but half the situation.

Graded exercise, all the way from massage and passive motions to walking varying distances with increasing grades, to golf, horseback riding, etc., has always been an important means of treating the convalescing or mildly decompensated cardiac. The underlying principle of all is the same, namely, that some work tends to increase the efficiency of the cardiac muscle and so of cardiac function and at the same time to improve the circulation in the peripheral vessels. This accomplished, more work will raise the function to a higher level and this increase should be continued so long as there is satisfactory functional response. Any thoughtful physician can plan for his patients adequate graded exercise by attention to these principles. Of course the mental reaction of the patient to his activity needs to be considered; too much solitary activity is usually a bad thing.

If the spirit of companionship, even of competition without actual physical competition, can be brought in, so much the better. Hence the excellent results obtained at "cures" and the good effects of limited games, which we are constantly observing.

It has always seemed to me that it is the principle of graded exercise for vascular system and body musculature that is the important thing rather than there being any particular virtue in special forms of baths, exercises, diet régimes and "cures" of various sorts.

Diets.—The convalescing cardiac requires a diet with sufficient calories to maintain his activity. If he is obese he needs a subcaloric diet with special restriction of carbohydrate until his weight is normal or slightly subnormal. Overeating at any single meal should be avoided. Moderate restriction of fluid intake is desirable; intake not to exceed 1,800 c.c. per twenty-four hours and preferably less. A normal balanced ration is best for nearly all, simply avoiding the obviously difficultly digested foods. Too much gaseous distention indicates carbohydrate reduction. Food fads flourish prodigiously in this country. Many of them certainly do no harm, but I can see no cardinal virtue in any of them for the cardiac.

Management When Symptoms are Absent or Slight

What to do when a cardiac lesion is discovered by physical examination and there have been no symptoms complained of or when slight symptoms, possibly of cardiac origin, have caused a patient to come for advice, often is the problem presented to the physician; to solve it satisfactorily is not always easy.

In the first place it is necessary to decide whether observed departures from strictly normal cardiac findings are to be taken as indicative of beginning cardiac disease or not. Often this decision requires the highest grade of clinical acumen. For example, certain murmurs, especially systolic ones, are known to be of no clinical import, having been continuously present, and so in evidence when there were no suggestions of cardiac symptoms. On the other hand, some severe cardiac conditions occur without any murmur being present. If a murmur, particularly a systolic murmur, is the only sign of cardiac lesion, it probably is of no import and at least should be tentatively so considered and the patient carefully observed over some period of time to make sure whether there are other evidences of cardiac disease. If with the murmur some cardiac enlargement is found, it is more probable that actual cardiac disease is present. Much the same attitude should be taken towards extrasystoles and palpitation. Precordial discomfort often is puzzling but on the whole more apt to indicate cardiac disturbance. The same may be said of slight breathlessness.

If these and other minor cardiac disturbances are decided to be evidences of early cardiac disease then definite treatment should be instituted. This, however, is by no means to say that digitalis should be given. In fact, in the great majority of patients it is not indicated. For these early evidences of cardiac disease, most important is advice as to occupation and exercise. For the sedentary exercise and especially out-of-door exercise such as walking, horseback-riding, golfing, etc., is indicated. For those working under conditions of physical strain, less vigorous pursuits are needed. In other words, there is an optimum level of physical activity for each cardiac and this may require much trial before it is found. Over-eating, too much smoking, alcoholic indulgence, etc., are to be guarded against. For the overweight, gradual reduction in weight by a proper diet is essential. Focal infections of all sorts, if actual, should receive proper treatment and be eradicated if possible. When symptoms increase, activities are to be curtailed in proportion. After all, it is the properly balanced life, guarded against excessive strain and protected from infections, which will longest maintain the individual free of embarrassing symptoms. To insist on too great reduction of activity perhaps is to err as badly as to advise too much. General nutrition and bodily and mental comfort are to be maintained as the best means of maintaining cardiac nutrition and function. Above all, do not frighten the cardiac case. All are familiar with the patient who, on the basis of a cardiac murmur, has lived long years in constant fear of sudden death. Restrictions can be made without unduly alarming the patient. If the patient shows definite evidence of cardiac disease by physical signs and yet has no symptoms that can be attributed to the cardiac disease, he should be managed as is described in the next section and warned against severe physical strains.

On the other hand, if symptoms are definite and appear to be increasing in severity, more active treatment should be instituted. For these a course of digitalis should be carried out, using the small divided dose method as already described. It is very well to put such patients to bed for a week or ten days of rest. Often rest in bed is all that is needed. Such patients need very careful testing out in regard to what activities they may safely undertake. It is well for them to report to their physician at frequent intervals for observation. When they report, it is particularly important to question the patient at these times as to the details of his daily life and the occurrence of any symptoms referable to cardiac insufficiency. Questioning is more useful than physical examination in this degree of cardiac disturbance.

Prevention of Cardiac Disease

Prevention in cardiac disease concerns itself with the prevention of those conditions likely to result in cardiac disease and, in case they occur prompt and thorough treatment to reduce, as far as possible, the probability of cardiac complication. Prevention and treatment of acute rheumatic fever is of greatest significance in this relation. Early tonsillectomy in the child who has had rheumatism should be carried out, if there is any sign of inflammation in the tonsils. Repeated tonsillar infections without rheumatism, too, indicate tonsillectomy. With development of rheumatic fever, a prolonged period in bed under the best of hygienic conditions (fresh air life and good nutritious food) is very important. Three months of such rest in bed is reasonable. This should be followed by frequent physical examinations, supervision of activities insuring sufficient amount of the proper sort of exercise, and continuous fresh air life with avoidance, as far as is feasible, of exposure to all kinds of infectious diseases. The occurrence of chorea is of even more significance than of rheumatic fever. Unfortunately methods intended to prevent cardiac involvement are less effective with chorea than with rheumatic fever.

As syphilis is a not infrequent cause of cardiovascular disease, this is an added reason in the management of the syphilitic for early, thorough and repeated courses of antisyphilitic treatment with mercury and arsphenamin. Focal infection should be eradicated promptly in the potential cardiac and streptococcic infections, in particular, should be promptly dealt with. Hypertension, when found, should receive prompt and thorough management for it is a not infrequent cause of chronic myocarditis. The same applies to arteriosclerosis though it is not so amenable to restrictive management as hypertension.

Hyperthyroidism frequently leads to chronic myocarditis and this is an additional reason for the prompt treatment of hyperthyroidism. It is well to remember that auricular fibrillation and other cardiac disturbances in hyperthyroidism are improved by operation and so should be regarded as a reason for prompt surgical treatment of the thyroid rather than an indication for delay in surgery. A few days of preoperative treatment with rest, iodin and digitalis reduces the risk of operation in these patients.

The flabby and fat, particularly if there are any slight cardiac symptoms, need dietary restrictions to bring about gradual weight reduction and graded exercises to tone up the flabby musculature.

TREATMENT OF SPECIAL CONDITIONS OCCURRING IN CHRONIC CARDIAC DISEASE

Auricular Fibrillation.—Auricular fibrillation in itself can, as an arhythmia interfering with diastolic filling of the heart, decrease systolic output and thus decrease cardiac efficiency. In addition, it occurs oftenest in hearts handicapped by valve lesion or myocardial disease. It is very frequent in patients with cardiac insufficiency and so its management is of great importance.

Auricular fibrillation can be treated in two ways, by digitalis and by quinidin sulphate. Digitalis acts chiefly by hindering conduction of many of the auricular impulses to the ventricles and so without correcting the auricular fibrillation removes most of its damaging effect on cardiac systolic output. Heart rate is slowed, heart rhythm remains unchanged. Also digitalis exerts its additional influence on ventricular muscle, etc. In auricular fibrillation digitalis is particularly efficient in improving cardiac function. Digitalis may be given in any of the ways already described.

Quinidin sulphate acts entirely differently. If effective, it converts the irregular rhythm of auricular fibrillation into a normal regular rhythm. According to Lewis auricular fibrillation is caused by the stimulus to contraction traveling in a sinuous circus or circle through the auricle in the region of the entrance of the superior vena cava, instead of the stimulus, as normally, being given off in a regular rhythm from the sino-auricular node. Quinidin sulphate acts on the heart muscle to slow conduction and increase the refractory period. If the latter action preponderates, the stimulus traveling in its circuit will reach a portion of muscle, previously stimulated, before it has recovered from the refractory period, and it cannot contract. This will block the path of the stimulus and in most instances under these conditions the sino-auricular node will begin to function and a regular rhythm will result. If slowing of conduction preponderates, the muscle will have recovered from the refractory period by the time the stimulus again arrives and fibrillation will continue. Actually about 50 per cent of patients with auricular fibrillation can be restored, at least temporarily, to normal rhythm by means of quinidin sulphate. Quinidin sulphate has no action on the heart muscle analogous to digitalis. Instead of a beneficial action there is evidence that quinidin is a heart muscle poison and so may decrease myocardial efficiency. So it would seem that its effect is limited entirely to influencing that phase of cardiac insufficiency resulting from the arhythmia. Consequently, improvement in cardiac efficiency from quinidin sulphate will depend on what proportion of the disability results from the arhythmia and what proportion results from valve defect or muscle lesion. If the former is relatively unimportant no improvement in cardiac function can be expected from quinidin.

Though, as already stated, about 50 per cent of patients with auricular fibrillation can be brought to a regular rhythm with quinidin sulphate, the practical results have been disappointing because relatively few of these patients are symptomatically better as the result for the reason given above; not infrequently the patient is quite unconscious of the change from fibrillation to regular rhythm when it does occur. Furthermore, there are unpleasant symptoms in some patients such as palpitation, headache, diarrhea, nausea, vomiting, abdominal pain and buzzing in the ears from the quinidin. Very often the regular rhythm, when attained, is not maintained. There are enough fatalities now reported to justify caution in using quinidin sulphate. An occasional patient with auricular fibrillation who does not do well on digitalis does better or is even in very good compensation on quinidin sulphate. Transient fibrillation sometimes is very annoying and may be prevented by quinidin sulphate. In my own personal experience results have been unsatisfactory, and I do not consider quinidin sulphate of very much real usefulness in treating auricular fibrillation. I would limit its use to those patients with auricular fibrillation before the stage of serious cardiac decompensation who do badly on digitalis, and to those patients who have transitory auricular fibrillation attended by marked discomfort. On this basis, very few cases would receive quinidin sulphate.

The method of giving quinidin sulphate is as follows: Give 0.2 gm. of quinidin sulphate in a capsule followed four hours later by another 0.2 gm. If there are no untoward symptoms indicating special sensitivity or idiosyncrasy to the drug, on the next day give 0.4 gm. three times a day, before meals, and continue until the rhythm becomes regular. After this, use a 0.1 to 0.2-gm. continuation dose to maintain a regular rhythm.

Larger dosage may be necessary to produce a regular rhythm but 2 gm. seems as much as should be given within twenty-four hours.

There is considerable difference of opinion as to whether or not to digitalize the patient before giving the course of quinidin sulphate. As digitalis and quinidin sulphate are rather antagonistic in action, it seems undesirable to give each drug at the same time. On the other hand, with evidences of considerable decompensation previous digitalization seems desirable and probably increases the number of patients whose rhythm is restored to normal.

It is to be remembered that quinidin sulphate therapy is still in a somewhat experimental stage. Already the first enthusiasm for its use has waned. To some patients it is definitely dangerous; to the majority with auricular fibrillation it works no great improvement even though it restores the rhythm to normal. In about half it will cause no change in rhythm or will produce a shift only to auricular flutter with a rate faster than that previously present.

Auricular Flutter.—Patients with auricular flutter should be digitalized by any of the methods already described. Under digitalis the flutter rhythm often changes to fibrillation and later normal rhythm is restored. The length of time digitalis should be given to these patients depends on the persistence of symptoms of decompensation. With return to normal rhythm, if digitalis is stopped, normal rhythm often continues until symptoms of decompensation recur.

Extrasystoles.—Extrasystoles are usually annoying; occasionally they hinder cardiac function; rarely they are a serious handicap to cardiac efficiency. Due at times to coffee, tea, tobacco, alcohol, masturbation or excessive sexual intercourse, discontinuance of the offender works a cure. At times they represent a fatigue phenomenon and are cured by rest, physical or mental or both. At times they follow infections and disappear with convalescence; their persistence is an added reason for prolonging the rest period following acute infections; obviously infection, particularly focal infections, should be eradicated as speedily and completely as possible.

Bromids are often very helpful in patients with palpitation and other annoying symptoms from or associated with extrasystoles. Quinidin sulphate seems to be useful for persisting annoying extrasystoles or those that interfere with cardiac efficiency. It is to be given as described under Auricular Fibrillation and has the same possible bad effects already mentioned. Here it is even more in an experimental stage than in auricular fibrillation.

Extrasystoles in the decompensated patient present a real problem for treatment; they may result from too much digitalis, particularly when causing pulsus bigeminus, and the digitalis should be discontinued; they are often present, even to producing bigeminy, before any digitalis is given. With few exceptions, these patients can be digitalized with great improvement of cardiac function and not infrequently with marked decrease or disappearance of the extrasystoles. In a rare patient, digitalis will increase these extrasystoles and the patient's condition is worse. In such cases the methods of treating cardiac decompensation, omitting the digitalis, are to be used.

Heart-block.—In cardiac decompensation heart-block does not change the method of treatment if the block is complete. Digitalis here is not contra-indicated for the possible digitalis effect on conduction no longer can occur and the other effects of digitalis may be expected. Sometimes the very slow rate of heart-block may be moderately increased by digitalis. However, complete block greatly decreases any expected benefit from digitalis. If conduction time is prolonged or partial block present, digitalis should not be given unless decompensation is marked and then it should be given cautiously by the small divided dose method.

Paroxysmal Tachycardia.—Paroxysmal tachycardia rarely occurs in patients with chronic cardiac disease. When it does occur pressure on the vagus nerve as it lies in the carotid sheath may stop the tachycardia. Strong pressure should be applied first on the left and then on the right side of the neck. Failing response to this, ocular pressure, that is, pressure on the eyeball, as strong as the patient will submit to, should be applied. Though painful this may stop the tachycardia when pressure over the vagus has failed. At times quinidin sulphate is effective. It may be used to end an attack or in small continuation doses to prevent attacks. The chronic cardiac disease otherwise should be treated as if paroxysmal tachycardia had not been present.

Pulsus Alternans.—Although pulsus alternans is indicative of an unfavorable prognosis, it does not indicate any change in the methods employed in the treatment of chronic cardiac disease. At times strikingly good digitalis effects are obtained when pulsus alternans is present (Fig. 4). Sometimes, but not often, pulsus alternans disappears under digitalis.

Chronic Myocarditis.—That chronic myocarditis contra-indicates the use of digitalis on the principle that a weakened myocardium should not be stimulated is a fairly generally held view. I can see no justification for this view and find that patients with chronic myocarditis form a group in which the general plan of treatment, as already outlined, yields very excellent results (Fig. 3, 4, 5, 6 and 7). It is in these patients when the pulse is not fast that many withhold digitalis, and yet in few conditions are better results obtained, notably a marked diuresis with disappearance of edema (Fig. 8).

High Blood-pressure.—It is often stated that one of the actions of digitalis is to cause vascular constriction and elevate the blood-pressure. This, though it may occur in animals, is not noted in cardiac patients unless the pressure is low from poor heart action. Certainly with high blood-pressure and cardiac decompensation digitalis does not increase the pressure. Cardiac disease with high blood-pressure should be treated in the same way as similar cardiac lesions in patients without high blood-pressure. The vasodilator group of drugs adds nothing to the efficiency of digitalis and the various methods already described.

Aortic Insufficiency.—Formerly it was thought inadvisable to use digitalis in patients with aortic insufficiency because the lengthened diastole resulting would permit of greater reflux of blood and a possible diastolic standstill from overdistention. This is no real danger and digitalis may be safely used in aortic insufficiency. In aortic insufficiency the mechanical effect of the valve lesion often plays a very important rôle in the cardiac decompensation and this is little, if at all, influenced by digitalis. Consequently aortic insufficiency cases do not, as a rule, give as satisfactory improvement as a result of treatment as do other valve lesions.

If aortic insufficiency results from syphilis, antisyphilitic treatment should be instituted, and in some cases there is marked symptomatic improvement, particularly in the pain and paroxysmal dyspnea which are present, probably from the accompanying syphilitic aortitis. In using arsphenamin in these patients smaller doses should be employed than used with other types of syphilis involvement, and particularly is a small initial dose indicated to prevent untoward results. A preliminary course of mercury is advised by many. With this and later arsphenamin in small doses (0.3 gm.) I have never seen any untoward effects.

Angina Pectoris.—If angina pectoris occurs with chronic cardiac decompensation it should receive the treatment elsewhere described for angina and in addition the management described in this chapter for the decompensated chronic cardiac case.

REFERENCES *

Carter. Further Observations on the Aberrant Electrocardiogram Associated with Sclerosis of Atrioventricular Bundle Branches and Their Terminal Arborizations; Clinical and Histologic Report of Case in Which Such Aberrant Complexes Were Obtained, Arch. Int. Med., xxii, 331, 1918.

Christian. Transient A-V Dissociation with Varying Ventricular Complexes Caused by Digitalis, Ibid., xvi, 341, 1915.

---

* This bibliography includes only relatively few of the great number of papers on the treatment of chronic cardiac disease. For the most part the ones included here are the papers published in the last few years and a few of the most important ones of an earlier date.

Christian. The Use of Digitalis in the Various Forms of Cardiac Arrhythmia, Boston Med. & S. Journ., clxxiii, 306, 1915.

——— Some Studies of a Diuretic (Theocin), Arch. Int. Med., xviii, 606, 1916.

——— Some Observations on the Use of Diuretics in Nephritis, Canad. Pract. & Rev., xlii, 269, 1917.

——— Chronic Myocarditis; a Clinical Study. Journ. Am. Med. Ass., lxx, 1909, 1918.

——— Digitalis Therapy; Satisfactory Effects in Cardiac Cases with Regular Pulse Rate, Am. Journ. Med. Sc., clvii, 593, 1919.

——— Chronic Myocarditis and Its Management, Southern Med. Journ., xiv, 587, 1921.

——— Right and Wrong Uses of Diuretics, Med. Clin. North America, iv, 1639, 1921.

——— Digitalis Effects in Chronic Cardiac Cases with Regular Rhythm in Contrast to Auricular Fibrillation, Med. Clin. North America, v, 1173, 1922; also Transactions Ass. Am. Physicians, xxxvii, 284, 1922.

——— Digitalis in Cardiac Disease. Boston Med. & Surg. Journ., clxxxvii, 47, 1922; also Journ. Iowa Med. Soc., xii, 307, 1922.

Clark-Kennedy. On the Therapeutic Value of Quinidine in the Treatment of Auricular Fibrillation, Quart. Journ. Med., xv, 279, 1922.

Cohn. Clinical and Electrocardiographic Studies on the Action of Digitalis, Journ. Am. Med. Ass., lxv, 1527, 1915.

——— The Clinical Pharmacology of Digitalis, Med. Clin. North America, i, 563, 1917.

Cohn, Fraser, and Jamieson. The Influence of Digitalis on the T-Wave of the Human Electrocardiogram, Journ. Exper. Med., xxi, 593, 1915.

Cushny. On the Action of Substances of the Digitalis Series on the Circulation in Mammals, Ibid., ii, 233, 1897.

——— The Therapeutics of Digitalis and Its Allies, Am. Journ. Med. Sc., cxli, 469, 1911.

——— Digitalis in Auricular Fibrillation, Journ. Pharmacol. & Exper. Therap., xi, 103, 1918.

Cushny, Marris, and Silverburg. The Action of Digitalis in Therapeutics, Heart, iv, 33, 1912.

Danielopolu. Action de la digitale sur la tension arterielle chez les hypertendus, Arch. d. mal. de cœur, x, 25, 1917.

Eggleston. Digitalis Dosage, Arch. Int. Med., xvi, 1, 1915.

——— The Influence of Large Doses of Digitalis and Digitoxin on the Blood-Pressure in Man, Journ. Am. Med. Ass., lxix, 951, 1917.

——— Administration of Digitalis by "Eggleston Method," Ibid., lxxiv, 733, 1920.

Eggleston. The Absorption of a Digitalis Body, Journ. Am. Med. Ass., lxxv, 463, 1920.

——— Some Newer Concepts in Digitalis Therapy, Am. Journ. Med. Sc., clx, 625, 1920.

Eggleston and Hatcher. The Emetic Action of the Digitalis Bodies, Journ. Am. Med. Ass., lx, 499, 1913.

Eggleston and Wyckoff. The Absorption of Digitalis in Man, Arch. Int. Med., xxx, 133, 1922.

Eyster and Fahr. Observations on the Use of Quinidine in Auricular Fibrillation, Ibid., xxix, 59, 1922.

Frey. Chinidin zur Bekämpfung der absoluten Herzunregelmässigkeit (Vorhofflimmern), Deutsches Arch. f. klin. Med., cxxxvi, 70, 1921.

Goodpasture. The Influence of Thyroid Products on the Production of Myocardial Necrosis, Journ. Exper. Med., xxxiv, 407, 1921.

Halsey. The Dosage and Clinical Value of Different Digitalis Drugs and Preparations, Southern Med. Journ., ix, 579, 677, 1916.

Hamburger and Priest. The Quinidine Treatment of Auricular Fibrillation, Journ. Am. Med. Ass., lxxix, 187, 1922.

Haskell, McCants, and Gardner. The Rate of Absorption of Various Digitalis Preparations from the Gastro-intestinal Tract, Arch. Int. Med., xviii, 235, 1916.

Hatcher. Digitalis Therapy and the Present Shortage of Drugs, Journ. Am. Med. Ass., lxix, 1524, 1917.

——— Some Observations on the Pharmacology of a Digitalis Body, Ibid., lxxv, 460, 1920.

——— Variation of Dosage Dependent on the Method of Administration, Ibid., lv, 746, 1910.

Hatcher and Brody. The Biological Standardization of Drugs, Am. Journ. Pharmacol., lxxxii, 360, 1910.

Hatcher and Bailey. The Clinical Use of Strophanthus, Ibid., lv, 1697, 1910.

Hatcher and Eggleston. The Emetic Action of the Digitalis Bodies, Journ. Pharmacol. & Exper. Therap., iv, 113, 1912.

——— The Stability of the Infusion of Digitalis, Journ. Am. Med. Ass., lxv, 1902, 1915.

——— Studies in Elimination of Certain Digitalis Bodies from the Animal Organism, Journ. Pharmacol. & Exper. Therap., xii, 405, 1919.

Hatcher and Weiss. The Seat of the Emetic Action of the Digitalis Bodies, Arch. Int. Med., xxix, 690, 1922.

Hay. Quinidine Sulphate in Cardiac Disease, Quart. Journ. Med., xv, 313, 1922.

Hewlett and Sweeney. The Quinidine Treatment of Auricular Fibrillation, Journ. Am. Med. Ass., lxxvii, 1793, 1921.

Kerr and Bender. Paroxysmal Ventricular Fibrillation with Cardiac Recovery in a Case of Auricular Fibrillation and Complete Heart Block While Under Quinidine Sulphate Therapy, Heart, ix, 269, 1922.

Korns. An Experimental and Clinical Study of Quinidine Sulphate: 1. Experimental, Arch. Int. Med., xxxi, 15, 1923. Ibid.

——— An Experimental and Clinical Study of Quinidine Sulphate, II. Clinical, Ibid., xxxi, 36, 1923.

Krehl. Zur Kenntniss des Digitalisgebrauchs und des Wasserwechsels, Deutsches Arch. f. klin. Med., cxxviii, 165, 1919.

Lawrence. The Effect of Digitalis on Blood-Pressure and Pulse-Pressure in the Presence of Cardiac Decompensation, Boston Med. & Surg. Journ., clxx, 37, 1914.

Levine. Auricular Fibrillation: Some Clinical Considerations, Am. Journ. Med. Sc., cliv, 43, 1917.

——— The Potency of Some French Digitalis Preparations, Boston Med. & Surg. Journ., clxxxii, 64, 1920.

Levine and Cunningham. The Margin of Safety of Intravenous Digitalis in Cats, Arch. Int. Med., xxvi, 293, 1920.

Levy. Restoration of the Normal Cardiac Mechanism in Auricular Fibrillation by Quinidine, Journ. Am. Med. Ass., lxxvi, 1289, 1921.

——— Some Results of Quinidine Treatment of Auricular Fibrillation, N. Y. State Journ. Med., xxii, 276, 1922.

Levy and Cullen. Deterioration of Crystalline Strophanthin in Aqueous Solution; Its Relation to Hydrogen Ion Concentration and a Method for Its Prevention, Journ. Exper. Med., xxxi, 267, 1920.

Lewis. Clinical Disorders of the Heart Beat, Shaw and Sons, London, 1912.

——— Clinical Electrocardiography, Shaw and Sons, London, 1913.

——— Action of Digitalis in Auricular Fibrillation, Am. Journ. Med. Sc., clxiv, 157, 1922.

——— The Value of Quinidine in Cases of Auricular Fibrillation and Methods of Studying the Clinical Reaction, Ibid., clxiii, 781, 1922.

——— Quinidine and Atropine in Fibrillation of Auricles; Clinical and Experimental Studies, Am. Journ. Med. Sc., clxiv, 1, 1922.

Lewis, Drury, Iliescu, and Wedd. Observations Relating to the Action of Quinidine upon the Dog's Heart; with Special Reference to Its Action on Clinical Fibrillation of Auricles, Heart, ix, 55, 1922.

——— Observations upon the Action of Certain Drugs upon Fibrillation of the Auricles, Ibid., ix, 207, 1922.

Lewis *et al.* Observations upon Flutter and Fibrillation, Ibid., vii, 127, 191, 247, 293, 1918-1920; viii, 37, 87, 141, 171, 193, 341, 361, 1921.

Marvin and White. Clinical Studies of Drugs of the "Digitalis Series." III. Apocynum and Convallaria, Journ. Am. Med. Ass., lxxvii, 1865, 1921.

Neuhof. Clinical Significance of Abnormally Wide Ventricular Deviation in Electro-Cardiogram, Arch. Int. Med., xxii, 45, 1918.

Oppenheimer and Mann. Clinical Experience with Quinidine in Auricular Fibrillation, Journ. Am. Med. Ass., lxxvii, 1800, 1921.

Oppenheimer and Rothschild. Electrocardiographic Changes Associated with Myocardial Involvement with Especial Reference to Prognosis, Ibid., lxix, 429, 1917.

Pardee. Notes on Digitalis Medication. 1. The Rate of Disappearance of Digitalis from the Body. 2. The Therapeutic Dose of Tincture of Digitalis, Ibid., lxxiii, 1822, 1919.

——— Rate of Absorption of Digitalis from Gastro-intestinal Tract, Ibid., lxxv, 1258, 1920.

——— Continued Use of Digitalis, N. Y. State Med. Journ., xxii, 131, 1922.

Pratt. Digitalis Therapy, Journ. Am. Med. Ass., lxxi, 618, 1918.

Pratt and Morrison. The Activity of American Digitalis, Ibid., lxxiii, 1606, 1919.

Robinson. The Significance of Abnormalities in Form of Electrocardiogram, Arch. Int. Med., xxiv, 422, 1919.

——— The Rapidity and Persistence of the Action of Digitalis on Hearts Showing Auricular Fibrillation, Am. Journ. Med. Sc., clix, 121, 1920.

——— The Value of Large Single Doses of Digitalis in the Treatment of Heart Disease, South. Med. Journ., xiii, 396, 1920.

——— The Therapeutic Use of Digitalis, Medicine, i, 1, 1922.

Robinson and Wilson. A Quantitative Study of the Effect of Digitalis on the Heart of the Cat, Journ. Pharmacol. & Exper. Therap., x, 491, 1918.

Roth. The Activity of Wild American Digitalis, Public Health Rep., xxxii, 377, 1917.

——— Digitalis Standardization. The Physiological Evaluation of Fat-free Digitalis and Commercial Digitalin, Bull. Hyg. Lab., No. 102, 1916.

Rowntree and Macht. The Standardization of Digitalis and the Potency of American-grown Digitalis, Journ. Am. Med. Ass., lxvi, 870, 1916.

St. Lawrence. Potential Cardiac Disease and Prevention of Organic Heart Disease in Children, Ibid., lxxviii, 947, 1922.

Strong and Wilmaers. The Potency of Some Common Digitalis and Strophanthus Preparations, Ibid., lxxx, 1308, 1923.

Weiss and Hatcher. Tincture of Digitalis and the Infusion in Therapeutics, Ibid., lxxvi, 508, 1921.

West and Pratt. Clinical Experience with a Standardized Dried Aqueous Extract of Digitalis, Ibid., lxxv, 77, 1920.

White. The Present Status of the Use of Drugs in Heart Disease with Especial Reference to Digitalis and Quinidine, Ibid., lxxix, 782, 1922.

White and Morris. The Eggleston Method of Administering Digitalis with Some Notes on Digitalis Lutea, Arch. Int. Med., xxi, 740, 1918.

White, Balboni, and Viko. Clinical Observations on the Digitalislike Action of Squill, Journ. Am. Med. Ass., lxxv, 971, 1920.

White, Marvin, and Burwell. The Action of Quinidine Sulphate in Heart Disease, to Abolish Circus Movement of Auricular Flutter and Fibrillation, Boston Med. & Surg. Journ., clxxxv, 647, 1921.

Wilson and Herrmann. Bundle Branch Block and Arborization Block, Arch. Int. Med., xxvi, 153, 1920.

Withering. An Account of Foxglove and Some of Its Medical Uses, with Practical Remarks on Dropsy and Other Diseases, Birmingham, 1785.

CHAPTER XII

ANGINA PECTORIS, CORONARY ARTERY DISEASE, CARDIAC PAIN

James B. Herrick

ANGINA PECTORIS

It is often said, and rightly, that angina pectoris is not a disease. It is really a group of symptoms with no one unifying pathologic lesion as an explanation. Acute and chronic syphilitic aortitis, atherosclerosis of the aorta and coronary arteries, coronary obstruction, acute pericarditis, acute myocarditis, neurotic and toxic states may cause these symptoms. It is, therefore, illogical to group them under a title that describes in reality but a peculiar type of pain. For the present, however, until a more rational method of classification is offered, this must be done. Already the approach to this desideratum is to be seen in the more accurate recognition of syphilitic aortitis, coronary obstruction, pericarditis and cardiac neurotic states. Ultimately the diagnosis will be that of the underlying disease and the pain will be regarded merely as a symptom or as a result, just as in the case of headache, where it is recognized that the primary trouble may be such diverse conditions as inflammation of the meninges, tumor, uremia or many other pathologic conditions. Such a conception of angina pectoris would make it much easier to describe treatment in a lucid and systematic manner.

Theories.—Various theories have been advanced to account for the syndrome of angina pectoris with its two outstanding features of pain and sudden death. Muscle spasm and muscle paralysis; a dilatation with tension on the cardiac nerves; neuralgia, hyperesthesia, a neuritis of the cardiac plexus; a change in the vasa vasorum, a spasm of the coronary artery; impairment of contractility of the muscle fibers of the heart, are among the explanations advanced. Osler has well grouped these various theories under four heads: neuralgia; cramp of the heart muscle or of parts of the same; weakness of the muscle with consequent dilatation resulting in tension and stretching of nerves; myocardial ischemia through coronary disease. To these possible causes must now be added the theory that the pain is due to tension on the nerves of the first portion of the aorta.

The two theories that to-day are most prevalent are those that make the changes in the coronary artery the important feature and those that ascribe to the aorta the predominant part. Ever since Jenner and Parry called attention to the marked changes in the coronary artery in cases of angina pectoris (illustrated in the famous case of John Hunter), pathologists and clinicians have ascribed an essential rôle to these arteries. In cases in which at autopsy no such change was to be found, a vascular spasm was assumed to have been present. The striking resemblance between the phenomena of intermittent claudication (thrombo-angiitis obliterans), as seen in the lower extremities, and the anginal attacks has been repeatedly pointed out. In each condition there is apparently until late enough blood for the part while it is relatively at rest. But upon increased muscular activity the obstructed arterial channel is unable to furnish the requisite amount and relative ischemia with pain results. From the time of Allan Burns, who more than a century ago advanced this explanation, to the present when physiologists are trying to imitate the condition by experiment, the "intermittent claudication of the heart" theory of angina pectoris has passed as the most nearly valid explanation of this serious malady.

However, some dissenting voices have always been heard, and especially of late has this view been vigorously challenged by Allbutt in England, Vaquez in France, and R. Schmidt in Germany. While admitting that diseased coronaries are often found, or even admitting that the diseased coronary may be the immediate cause of death, these writers declare that the essential lesion is aortic. If at the autopsy attention is not too much focussed on the coronary, quite uniformly there will be found lesions in the first portion of the aorta, generally atheromatous or syphilitic, with changes to be made out especially in the middle and outer coats of the vessel. These, it is contended, are the lesions of pain; the coronary lesions are concomitant, secondary or complicating, though perhaps fatal. On exertion, as on walking, or under psychic urge the heart increases its work, the blood-pressure rises, the supra-sigmoid aorta stretches, the rich network of highly sensitive nerves in the diseased middle and outer coats is thus irritated and conveys to the cord segments and the brain the impression of an exaggerated irritation, and the substernal pain is the result; in many instances, through the intensity of the irritant, there is an involvement of neighboring segments and a more extensive reflection of pain, as to the neck or arm.

So strongly are these views held that Allbutt says: "For me the coronary and myocardial hypotheses are dead and buried." Manouelian says: "The coronary theory of the syndrome has fallen to pieces before the rigorous observation of clinico-pathologic and physiologic facts."

Vaquez contends that the sudden distention of the diseased aorta is the cause of the attack and claims to be able to show during such an attack not only an increase in blood-pressure but by physical examination an actual enlargement of the aorta which disappears as the attack subsides. R. Schmidt says that at autopsy the findings in the coronary artery are variable but atheroma of the ascending aorta of the arch is found with great constancy.

It is not necessary in an article on treatment to discuss more fully the theories as to causation. Yet it must be added that the question is by no means settled. Perhaps there is truth in either hypothesis. It is certain that a sudden coronary obstruction may cause an attack that so far as pain is concerned often resembles that of the typical paroxysmal angina; the aorta need not be diseased as some autopsies have shown. On the other hand there are many autopsy reports on patients with typical anginal seizures where the surprise has been the extensive aortic change with the lack of coronary and myocardial disease. Even Vaquez and Lutembacher are now speaking of two types of angina, one the ordinary "angina of effort," and the other an angina due to ventricular distention,—that is, myocardial weakness, reminiscent of Mackenzie's muscular exhaustion theory,—which they call decubital angina because it is apt to occur with no effort and with the patient in bed. The pain, however, they still regard as periaortic as the irritant travels the same path as though it had primarily originated in the diseased periaortic region.

Symptoms and Diagnosis.—The essential clinical features of angina pectoris are too well known to warrant repetition. There still prevails among physicians more or less diversity of opinion as to what should be called angina pectoris, and of necessity there must to-day remain much confusion in the interpretation of the clinical entity—a complex of symptoms or syndrome some call it—concerning whose underlying pathogenesis there is still so much uncertainty. Most of these mistaken notions have arisen from too strict adherence to the picture presented by the typical case in which, on walking, an excruciating pain radiating to the neck and arm brings the patient to a sudden standstill in an agony of physical and mental suffering as he feels himself face to face with death. But we are no more justified in drawing the outline of angina and arbitrarily saying that any alteration of this picture by the addition or omission of any line is not allowed than we are in dogmatically stating that lightning pains, Argyll-Robertson pupil, with loss of knee-jerk, alone make the picture of tabes, or that the grouping of symptoms in Basedow's disease is unalterably fixed. In other words, variations from this type are encountered.

Anginal attacks, even fatal attacks, may take place in one whose blood-pressure is not high, whose peripheral arteries give no evidence of sclerosis, and whose heart is not demonstrably enlarged. Isolated coronary sclerosis or an aortitis, particularly a syphilitic process at the very beginning of the aorta, may be the underlying cause of the angina, and the general arteriosclerosis so commonly and correctly associated in the physician's mind with this disease may be lacking.

Another not uncommon mistaken notion concerns the number, duration and severity of the attacks. So frequently are the attacks single or very few, of extreme severity, and lasting but a few seconds, that this conception of the disease is indelibly fixed in the mind of the practitioner, and shuts out from his consideration any possible marked variation from this type as consistent with true angina. As a matter of fact, the attack may be solitary, the one being fatal; or the attacks may be several, at long intervals, or they may be frequent, many in a day. I have seen as many as six and eight paroxysms in a day. Neusser mentions a woman with twenty a day. They may vary in degree from those almost unbearable to mild, almost negligible, pain. And *angina sine dolore* is undoubtedly a reality. Some of the attacks may last much longer than a few seconds or a few minutes, and there is apt to be left a soreness or ache in the shoulder, elbow or inner side of the left arm, perhaps an ache or numbness extending down to the fingers. This residual pain may carelessly be regarded as rheumatic or neuritic. The tenderness of the skin over the left chest, as described by Head, Mackenzie and others, may be present.

It is not to be forgotten that the usual exciting causes, overexertion, brisk walking, overdistended stomach, fit of anger, etc., may be lacking. And the cause, for example, a rapid walk, that brings on an attack to-day may to-morrow fail to do so. Charles Sumner at times had an attack from slight movement, such as turning in his revolving library chair, while he might be free when engaged in earnest debate, though making vigorous gestures. And, again, one must be prepared for true angina in women; probably 3 to 10 per cent of the cases. This fact should be kept in mind even though there is plainly to be made out a strong neurotic taint, for the clearly functional, even hysterical, manifestations may easily mask the more serious organic condition that underlies.

Rarely unconsciousness occurs. Nearly always the patient stands or sits still, immobility and the unwillingness to lie down being quite characteristic. Yet some patients are active during the seizures and walk about while suffering excruciating pain. In one instance I have known the patient, an old colored man, to lie flat—on the floor, the sidewalk or the porch—wherever the pain caught him, and to insist that only in this position was the pain endurable.

Prognosis.—Angina pectoris is truly a most formidable condition as regards its outlook. Real recovery is rare. Patients may, however, live many years, even twenty, as did John Hunter. I have patients who have lived ten years since the first seizure. On the contrary, the first attack may be fatal. Of a series of 200 cases the average duration of the disease in 50 whose deaths were reported was nearly three years from the onset of the symptoms. One patient lived seven years, having very few attacks. Of these 50, 44 per cent died between the ages of fifty and sixty and 82 per cent between forty and seventy years. This corresponds with the incidence of the condition. Of 200 patients the onset was between forty-one and seventy years in 82 per cent. We know of no way to foretell how long a man with angina may live. Rapidly recurring seizures, signs of cardiac failure and complications will naturally make the prognosis grave. If so-called pseudo-angina is included, the prognosis will be much better.

Prophylaxis.—How much is accomplished in the way of warding off angina pectoris can never be determined in a particular case. However, in those whose family history shows early deaths from cardiovascular disease, and especially sudden death from angina; in those who have evidence of beginning hypertension; in the syphilitic, and those with histories of rheumatic and other infections liable to recur, directions as to right living may, if heeded, accomplish something in the way of retarding the advance of sclerotic or chronic inflammatory changes in vessels or myocardium, and may postpone the appearance of angina. Removal of foci of infection in tonsils, teeth, gall-bladder, urinary bladder, etc., early and vigorous treatment of syphilis, especially syphilitic aortitis, may insure future freedom from this dread disease. In patients who may be regarded as "threatened" with angina pectoris, the physician may be doing more by timely advice as to moderation in diet, overwork, and worry than by later administration of drugs.

TREATMENT

Rational treatment is based on a knowledge of etiology, pathology, disturbance of function and the natural course of a disease. In many cases of angina pectoris the etiology is obscure; as has been shown, there are conflicting views as to the underlying pathologic anatomy; the heart may seem functionally perfect except as regards the abnormal appearance of pain; the tendency of the disease is commonly to progress toward a fatal termination, often a sudden death. No wonder then that by lay people and by physicians the treatment of the condition is so often regarded as useless or at best as purely symptomatic, formal, and promising little or nothing in the way of cure or even improvement. As a rule, also, the patient comes for help at a time when cardiovascular changes are advanced. Pronounced arteriosclerosis, hypertension, cardiac hypertrophy, old luetic lesions, valvular disease with beginning myocardial failure, are discouraging features with which to confront the physician. He recognizes that the painful seizures are but one manifestation of long-standing processes, the removal of which is beyond his power. Yet in spite of these depressing features the treatment is not without some reward in the way of palliation of distressing symptoms, prolongation of life, and at times a restoration to comparatively good health.

Active Treatment.—Assuming that the physician has made his diagnosis of angina pectoris, what shall he say to the patient as to the nature of his malady? Is the patient to be told frankly that he is a sufferer from angina pectoris, "neuralgia of the heart," and allowed to leave the physician's office with the horror of a sudden death forever after hanging over him, to say nothing of the thought of repeated attacks of the terrible pain; or is he to be reassured, the real nature of his illness concealed from him and his fears allayed by dwelling upon "indigestion," "nervousness," "neuritis," etc.?

Recently I ran across two statements that fairly well illustrate the diametrically opposed practice of many physicians regarding this disease. In a meeting of a state medical society in 1922 a well-known physician from a large city is reported to have said: "My own method for years has been to tell these patients plainly that they may die suddenly, to make their wills, and have no further responsibility requiring anything of that kind, so that no unusual loss will fall on their families." Another physician, likewise from a large city, in a state medical meeting in 1919, said: "I make a practice of telling my patients frankly, lying cheerfully, that they are going to get better . . . minimizing the gravity of the disease." Now I do not believe the reports of these discussions fairly represent the actual practice of either of these doctors. If we are obliged to choose between cheerfully lying and promising improvement or recovery, and bluntly telling a man at once to make his will, we must unhesitatingly decide in favor of the latter plan. Lying in medicine, even though practiced with the laudable object of helping a patient, is not only dishonest but it is a poor policy. In the end it generally works to the harm of the patient and reacts disastrously upon the physician. What we should aim to do is, without lying, to convey to the patient as correct and complete a notion of his ailment as he can comprehend and to speak as hopefully of the future as the facts warrant. To send a man with angina from the office with only the dark side presented—the recurrence of attacks, the sudden death, the necessity of making the will—is to rob him of a hope to which he is entitled, and is in reality a misrepresentation or a lying, if you please, as reprehensible as to promise a cure where cure is improbable. Recovery has been seen, improvement is common, and the patient should not be depresssed by an absolutely hopeless prognosis. As Osler has somewhere well said the physician may properly dwell upon the case of John Hunter, who lived for twenty years, rather than on the case of Arnold of Rugby, who died in an early attack. To explain too much in detail to a patient not intelligent enough to understand is often more harmful than to speak dogmatically as to what to do without advancing reasons. On the other hand, to omit reference to the serious nature of the illness, to minimize its gravity too much, is liable to lead to carelessness on the part of the patient as to habits of living, and may cause unfortunate results. And the patient will soon lose confidence in the physician who proves himself unable to relieve what he has called "indigestion" or other trifling ailment. Unusual tact is required of the physician who handles these cases, for he must tell the truth, but tell it in such a way as not harmfully or unnecessarily to alarm the patient, and yet so as to insure his coöperation in the proper carrying out of the details of the management of the case. The doctor who advises the patient to make his will does it, we are sure, not in the blunt, almost brutal way implied by the reported remarks but in a tactful manner giving a minimum of alarm, and we know he sends his patients from the office with the feeling that the illness, even though serious, is not without its element of hope.

After all there is no routine practice in this respect. No two doctors will tell the same facts in the same way. One must talk to one patient in one way, to another in another. One must individualize in the treatment of angina pectoris as in the treatment of any other disease, and in no respect more than in telling the patient the nature of his illness, the outlook, and the manner of its management.

To the friends and relatives there should be the utmost frankness of expression as to the probable outcome and the necessity of strict obedience to orders. This is necessary as a protection to the doctor who, in the event of sudden and unpredicted death, may be blamed for not having understood the case, and also in order to obtain the intelligent coöperation of the friends. And there should not be delay in speaking to friends. I have had a patient with angina drop dead thirty minutes after the first office consultation and before such warning to friends had or could have been given.

Treatment during an Attack.—Most attacks of the paroxysmal minor type subside spontaneously when the patient stops involuntarily and remains sitting or standing for a few minutes. One who suffers in this way learns to slow up or stop at the first hint of substernal constriction and in this way wards off many seizures.

The nitrites have a deserved reputation for being helpful in preventing or shortening the duration of the attacks. Many patients rely on prompt inhalation of nitrite of amyl which they carry in the shape of the glass pearls, each containing a few drops of the remedy. One of these pearls may be crushed in the handkerchief and the nitrite inhaled. Often within a few seconds the sense of constriction and the pain disappear and the patient is able to move on slowly. Pills or tablets of nitroglycerin gr. 1/100 may be conveniently carried in the vest pocket and may be used instead of the amyl nitrite. The pill or tablet should be chewed and held in the mouth some time before being swallowed. Fresh supplies of pills should be obtained from time to time, as they may have deteriorated if too old. Many physicians in order to avoid this objection to the pills have their patients carry with them a small bottle of spirits of glonoin. One or 2 drops placed on the tongue may be substituted for the pill or tablet. In case a single dose of nitroglycerin does not relieve the pain, a second one may be taken in a few minutes; or even a third. Some patients learn to use 1/50 gr. at the start. So far as known, no harm comes from continuing the use of the nitrites during the attacks over a period of months or years. The physician must not rest his faith too implicitly on the efficacy of the nitrites. In many cases the effects are almost marvelous in their prompt relief of distress. But in other instances, and one cannot always tell why, no relief is forthcoming, though the pounding in the head, the flushing of the face, the congestion of the eyes give evidence that the physiologic effects of the drug are manifest. So disagreeable may be these effects that the patient in the absence of relief of pain soon gives up the use of the remedy.

In severer attacks that may be attended by greater suffering, and with the pain persisting for a longer time, if the nitrites are ineffectual morphin may be necessary. This should be given hypodermically and little can be expected from a dose smaller than ¼ gr. Chloroform is said by some to be of use as a last resort. Many, perhaps most, of the cases in which large doses of morphin or chloroform are necessary, because of the long continuance of very severe pain, are instances of coronary obstruction, generally by a thrombus. These will be described later.

A large mustard plaster enveloping the chest is advised by Martinet. I have had no experience in its use or in the employment of other counterirritants. If there is acute edema of the lung and a dilated heart, he advocates bleeding as does Lutembacher in certain cases. Some of these are instances of coronary obstruction. In ordinary anginal attacks bleeding is unnecessary. It may possibly have a place where blood-pressure is very high or where, as Martinet says, there is edema of the lung with dilated heart.

Treatment in the Intervals between Attacks.—One should guard against managing a case of angina pectoris according to some routine, machinelike, fixed plan. The causes that induce individual attacks may differ, the clinical manifestations are not always identical and the underlying etiologic, anatomic and pathologic conditions may greatly vary in degree and in character. All these factors should be carefully studied. Treatment should be instituted, having for its object the elimination, so far as possible, of the basic abnormal conditions that lead to serious and life-threatening disturbance of function. If this cannot be accomplished the attempt should be made to minimize the harmful effects of these lesions. For the obese, gouty individual a different régime should be prescribed from that recommended to the syphilitic with an aortitis. The aged, sedentary, atheromatous man with failing myocardium may need treatment other than that advised for the energetic nephritic with hypertension. The neurotic individual who suffers from so-called pseudo-angina is to be handled in still another manner. In other words, individualization is essential.

The provocative cause must, so far as possible, be avoided. Rapid walking, especially up hill or against a strong wind, must be interdicted. Especial caution is necessary after heavy meals. Many patients who are able to walk quite freely at other times find that after the heavy evening meal the painful seizures are readily provoked. Rest after such meals is often very helpful.

Patients should be warned against permitting themselves to become angry or greatly excited, as such emotional disturbances are in some patients a frequent cause of the pain. Sexual excesses must be avoided. Tobacco in large amounts is generally believed to be a cause favoring the outbreak of anginal pain. The physician must in a given case use his judgment as to cutting off entirely the use of tobacco as well as of alcohol and coffee. At times it is certainly wiser to allow them in moderation than by suddenly removing them to upset a patient who has used them habitually for years.

In general, everything in the nature of excess is to be avoided. The amount of exercise is to be determined largely by the effect. Just how far a particular patient may be allowed to walk, or how rapidly, can be determined better by the patient than by the doctor. He should stop short of the point where the earliest symptom of pain, constriction or numbness indicates to him the beginning of angina. He knows by experience about what is the measure of his capacity in this respect. It is a mistake to condemn these individuals to confinement in bed, or even in the house, except in very aggravated cases, or when accompanying conditions, such as myocardial weakness, make such rest imperative. In fact moderate exercise is sometimes beneficial. Carefully graduated exercise may be helpful in a failing myocardium. I have known many sufferers from angina who have not only been able to continue to play golf but who have been benefited by the temperate indulgence in this sport. When exercise is impossible, mild massage may be substituted.

The strenuous life may need to be toned down. The man of business who works long hours carrying heavy burdens of responsibility and worry may have to make radical changes in his habits. He must let others do some of his tasks in order to lighten his own load. He must be told plainly that he must not put more irons into the fire but must perhaps take out some. And he must slow up, must not exceed the speed limit, etc. The symptomatic improvement in some patients of this kind when they are relieved of nervous and physical overstrain is very marked indeed. And at times, too, there is a gratifying reduction in blood-pressure, less nervousness and insomnia, better digestion. More frequent and longer vacations when these are possible are often followed by improvement. A trip to the South in winter may be of great benefit.

The matter of diet has to be carefully considered. The close relation between anginal spells and an overloaded stomach is often noted by patients and is commented upon by many clinicians. Especial attention is needed when the patient is plethoric, obese, has high blood-pressure, is constipated, flatulent, when he is a heavy eater or has nephritis, diabetes or gout. Many an anginal patient eats too much and too rapidly or eats improper food. Simple diet with an omission of the richer articles, such as rich stews, gravies, dressings, salads, soups; rich sweets, such as cakes, puddings, pies, pastries, fried and greasy foods, will tend to lessen the amount of gas in the stomach and bowels, reduce the load of food in the stomach and cause decrease in weight. Smaller amounts or starchy foods and of fats may be advisable. Meat once a day is generally enough. And as a rule, smaller meals as to quantity are in order. If the patient feels the need of more food when he is taking very small meals, a light lunch may be taken between the regular meals. The benefit from cutting down the amount of food and reducing in weight is sometimes so marked as to lead the patient to go to an undesirable extreme. I have known a man to be so relieved of his evening attacks of pain by the simple process of eating a very light supper that he voluntarily gave up this meal entirely, going without eating from noon of one day to seven in the morning of the following day. This was unwise as he began to show evidence of general and cardiac weakness owing to his too prolonged fast.

Constipation can generally be regulated by diet in which emphasis is placed on cooked and raw fruits, vegetables, coarse cereals, butter and cream. Where possible such regulation of the bowels by diet is better than the use of cathartics. An occasional laxative such as a morning saline or an evening dose of cascara, senna or phenolphthalein may be necessary.

It may be mentioned that the expulsion of a large amount of gas from the stomach does not always indicate indigestion. Some of these patients are aërophages; they swallow air, unconsciously of course, and think the whole trouble is due to a deranged stomach. Such individuals can usually be broken of this habit if its nature is fully explained to them. Drugs do little good. Suggestive treatment or psychotherapy is of much more value.

I have spoken of the necessity of suiting the treatment to the individual case. This applies to diet as well as to drugs. The obese, the diabetic, the gouty, or the nephritic patient who suffers also from angina may need modification of the diet to suit his particular case. But the general principle of simplicity and moderate amount of food at a meal should hold in these special cases.

The question of the advisability of prolonged rest in bed is often raised; as a routine treatment it is unnecessary. If there are signs of cardiac weakness such as marked dyspnea, râles, cyanosis or edema, such rest is in order until muscular tone is reëstablished. A nervously exhausted patient may also be helped in this way. At times such rest gives the doctor a better opportunity to study the patient, to get his confidence, allay his fears and to start him on a strict regulation of diet, than seeing the patient only at broken intervals in the office.

Drugs.—There is no drug that has anything like a specific action in angina pectoris. Reference has already been made to the use of the nitrites during an acute attack. Their long-continued employment in the intervals, even in cases where there is a high blood-pressure or where one may reasonably assume the existence of sclerotic coronaries, is seldom followed by improvement. The nitrites of sodium or potassium or erythrol tetranitrate are the preparations advocated by those who give the nitrites in this way. There are those who believe their long-continued use tends to harmful weakening of the heart's power.

The two drugs that seem to do the most good are iodid of potassium (or sodium) and digitalis. That subjective improvement often follows the use of iodids there can be no doubt. Just how they act and in what class of cases the benefit is to be seen cannot be definitely stated. Even in non-syphilitics they may be helpful. By this is not meant that they will always do good. In many instances, good results are not seen. A saturated solution of potassium iodid may be prescribed, and from 10 to 30 drops in one-half glass of water taken after or between meals. Iodism and a sensitive stomach occasionally necessitate stopping the remedy. It should be given, on and off, for long periods (months) in order to get the best results.

Digitalis is indicated whenever there is evidence of a failing heart. If the patient with angina has dyspnea, cyanosis, râles and cough, or edema, all clearly due to myocardial weakness, digitalis should be given. There may or may not be concomitant valvular disease, adhesive pericarditis, or evidence of myocardial fibrosis; cardiac irregularities such as extrasystoles or auricular fibrillation may or may not be present; the heart may be altered in size or shape or may be normal in these respects.

But if there is evidence of weak heart digitalis is in order. The notion that the existence of angina pectoris contra-indicates the use of this drug is not well founded. Also in cases of hypertension or of cardiac hypertrophy in nephritis, when cardiac asthma appears, perhaps with alternating pulse or gallop rhythm, neither the fact of the high blood-pressure nor the associated angina pectoris should lead to the withholding of digitalis. How the drug acts may be questioned, but that it often helps in this type of case is certain. Any of the reliable preparations may be employed exactly as in failing heart with valvular disease. Large doses may be given for a few days and then smaller ones. It may be necessary to continue the use of the drug for long periods. There is apparently no harm in this if small doses, for example, 10 to 20 drops of the tincture, are given.

Many other remedies have been advised. Not a few physicians use sodium theobromin salicylate in doses of 3 to 10 gr. They give this for its supposed power of dilating the coronary artery and for its diuretic effect. It also tends to strengthen the heart's action. Combinations of this and similar drugs with iodin have been advised in the shape of proprietary remedies such as ioddiuretal (Askanazy) and eusterin (von Noorden). Euphillin has some warm advocates.

When a patient with angina is proved to be syphilitic and when syphilis may reasonably be looked upon as the underlying cause, treatment should be directed along these lines. Here arsphenamin or any of its modifications may be used but with extreme caution. Ehrlich's warnings, the knowledge of the Herxheimer reaction and the unfortunate experiences of many physicians should teach us not to use the drug in these cases, or to employ it only in small doses. A weakening of the wall of an aneurysm or of a syphilitic aorta or a swelling of a lesion in the coronary may be followed by serious or fatal consequences. It is better in general in these cases to rely on iodids and mercury. Here the iodid may be given in gradually increasing doses and mercury may be administered in any manner desired. Results in some instances are very satisfactory. A safe procedure in a case of angina clearly due to syphilis, as in an aortitis or an aneurysm, is to give first mercury, then iodid and lastly, if necessary, arsphenamin.

Surgery has been employed for the relief of angina pectoris, that is, for relief from the pain. Jonnescu found that by cutting the centripetal path of the impulse from the heart he prevented the recognition as pain of any irritant in the heart or aorta. By operation he cut the cervical ganglia with good result, so far as relief from pain was concerned. Recent articles by Coffey and Brown, Cutler and others report successful operation in a few cases. Only the future will show the possibilities and advantages of surgery in this condition and determine just what part it may play in treatment. In some cases it may relieve unusually distressing symptoms and may make life more endurable, restoring some of a man's activity by the removal of the paroxysms of pain. It may thus be a justifiable procedure even though it leaves untouched what is commonly regarded as the underlying pathologic condition in the aorta and coronary arteries. Care should be exercised in selecting these cases, for in many who have angina with the attendant badly diseased cardiovascular systems an operation must be regarded as more or less dangerous. Wenckebach's judicial estimate of the procedure, that comes as this article is going to press, is well worth consulting by one who contemplates advising operation in cases of angina pectoris.

There are many other drugs and remedial measures that have been advised in the treatment of this condition. Among these may be mentioned the intravenous injection of glucose solutions, the use of X-ray and radium, blisters to the precordium, etc. There is too little evidence, however, either theoretical or practical, to warrant discussion of their merits.

Pseudo-Angina Pectoris

Little has been said of false or spurious angina, the so-called pseudo-angina. Many object to the use of the term, and with some reason. But by whatever name it is called, it must be recognized that there is a type of precordial distress and pain often closely resembling that just considered but where no underlying organic lesion exists in the aorta, coronary or myocardium. Possibly in some instances coronary spasm may be at fault, as Nothnagel and others contended. In some cases toxic substances, for example, tobacco, seem to induce the pain. But in nearly all of these cases there are psychic and nervous influences at work. Nervous exhaustion, as Hay says, may lower the threshold to pain. If these factors are removed the attacks cease.

It is not an easy task to decide as to the purely nervous origin of a pain in the heart region. One must often withhold judgment and for a time be on the safe side and consider the case as possibly genuinely organic in character. The main distinguishing points are the neurotic make-up of the patient, often a woman, the frequency with which psychic or emotional causes bring on an attack—worry, anxiety, domestic disagreement—and the fact that the attacks often occur while the patient is quiet or in bed. The attacks may last much longer and are apt to vary in character, pain being described at one time as in the upper chest, at another in the submammary region, in the arm, neck, head, "all over the body." The patient is often restless, tossing about in bed, moaning, presenting a very different picture from the immobile, statuesque figure of the real angina. Often there are no cardiovascular changes to be made out. If there are cardiac lesions with the neurotic feature added, the neurotic patient, much as in a case of traumatic neurosis, involuntarily fixes attention on the diseased organ and the physician's diagnostic acumen is taxed to the utmost to decide as to the real nature of the pain.

Treatment.—The treatment of this nervous type is largely a question of psychic management. No hard and fast rules can be given. Relief from bodily and nervous fatigue and from cares and worries is sometimes all that is needed. Many patients have had heart disease suggested to them by the doctor or by friends, perhaps some member of the family who has recently died had been a sufferer from angina or other painful heart affection. A careful examination, an assurance that there is no real cardiac affection, patience and persistence will generally clear up these cases. Drugs are seldom needed. Occasionally bromids, a laxative, or iron for an anemia will be helpful.

ACUTE CORONARY OBSTRUCTION

There is a type of cardiac pain, due to sudden obstruction of a coronary artery or some of the larger branches, that may appropriately be discussed under the caption of angina pectoris.

Acute obstruction of any large branch of a coronary artery was for a long time regarded as nearly always a suddenly fatal accident. This view was fostered largely by the conception that the coronary arteries were terminal arteries with only negligible anastomoses. An obstruction as by a thrombus would then result in an anemia of a large area of the heart muscle, which area would become functionless and death would be the natural inevitable result. Cohnheim's experiments in ligating the coronaries in dogs seemed to bear out this notion. But later investigations by anatomists, experimenters, pathologists and clinicians showed that there were rather free and clearly functioning anastomoses between the right and left coronaries, that many experimental animals lived though trunks of considerable size were ligated; that autopsies on human beings showed obstructive lesions that were clearly of long standing, and that patients who had symptoms suggestive of coronary obstruction sometimes lived for a considerable time and were found later at autopsy to have had the lesion suspected during life. The conclusion of all this is that there must be a time at which the obstruction occurs when symptoms are present that might permit us to recognize these cases. Some years ago I made a tentative grouping of these cases into (1) those in which death is instantaneous; (2) those in which death is sudden, occurring in a few minutes or a few hours from the time of the accident, the patient being practically moribund when seen by the physician; (3) a hypothetical group where obstructions in small branches produce slight symptoms only, not sufficient to induce the patient to appeal to the doctor for help, and (4) a group in which a branch of considerable size is obstructed, severe symptoms are produced, death often following after several hours or days; however, not a few patients in this group recover, with the heart restored partially or completely to its functional integrity. It is with this fourth group alone that we are concerned from the standpoint of treatment. The patients in the first two groups are beyond help, those in group 3 do not need it. It goes without saying that any such classification is quite artificial and that the groups shade off into each other with no clear line of separation.

As a basis for the thrombotic obstruction—and this is the common lesion—there is found with great uniformity a sclerotic coronary artery with resulting roughening of the intima and narrowing of the lumen. An area of softened, infarcted myocardium is the result, slight or extensive, depending on the size of the vessel occluded and the degree of collateral circulation. If the infarct extends to the outer surface of the heart there will be a roughening of the pericardium, an "aseptic pericarditis." Often the lesion is more marked in the subendocardial tissue. A favorite site for these infarcts is near the apex of the left ventricle, the descending branch of the left coronary being the artery obstructed.

Patients of either sex are affected, men oftener than women. They are nearly all adults or the aged. Vascular disease may or may not be present in the peripheral arteries or manifest by heightened blood-pressure, enlargement of heart, or cardiac irregularities. Paroxysmal angina pectoris has been present in many of the patients.

When the obstruction occurs there is commonly severe, sudden pain in the sternal, mammary or upper epigastric region. It may come on during sleep, or without any of the usual causes, such as effort. The pain is of unusual severity and generally lasts longer than the ordinary anginal seizures. "The worst attack I have ever had" is a common description. It may or may not radiate to the arms and neck. It may radiate to the epigastrium. If vomiting occurs and if, as is common, the patient shows signs of shock or collapse in the ashy color of the face, the cold sweat, the rapid small pulse, the resemblance to a subphrenic accident may be very striking. Great care has to be exercised to exclude such conditions as perforated gastric ulcer, perforated gallbladder or acute pancreatitis. There may be dyspnea, râles may appear in the chest, and the patient may be cyanotic. The pulse is commonly rapid and at times irregular from extrasystoles or fibrillation. In some cases I have found it rather slow and fairly regular. The heart tones are commonly feeble—sometimes startlingly so. Blood-pressure falls. Strange variations from day to day may be noted in the rate and rhythm of the pulse and in the blood-pressure. Occasionally a pericardial friction is made out over the infarcted area. An acute emphysema of the lungs sometimes produces a confusing note of hyperresonance over the chest. The mind may be clear, but is sometimes clouded. Delirium is sometimes seen. The temperature may remain normal throughout or it may rise even to 102° F.

If the patient survive more than a few hours or days the pain may entirely disappear or it may recur at irregular intervals with even the slightest movement in bed or with no movement at all. A status anginosus may develop. The heart may dilate, a systolic murmur appearing over the mitral area. The liver may become engorged, the legs become edematous, the albuminous urine of congested kidney appear.

Many of these patients die after hours, days or weeks. Some make a quite rapid recovery in a few weeks. Others after months are able to be about but with hearts muscularly weak, with action irregular. The extent to which the heart is damaged depends naturally on the location and size of the infarct, and the degree to which function can be restored by collateral circulation. Electrocardiographic studies show altered tracings, many of them suggestive of injury to the muscle and conduction system. Some help is obtained from the electrocardiograph in diagnosing these lesions and in estimating the amount and location of the injury but the subject has not yet been worked out enough in detail to permit of very accurate statements.

The important fact to remember is to think of this accident when confronted by these peculiar cases of "indigestion," "ptomaine poisoning" or "decubital angina" of the French. The condition is not merely a postmortem curiosity as it has been at times called, it is a condition permitting of a reasonably accurate diagnosis in a goodly proportion of cases. And a diagnosis carries with it a prognosis and an appropriate treatment. The prognosis must be neither the fatal prognosis that formerly attached to coronary obstruction nor the favorable one that goes with "acute indigestion." The condition is always of serious import yet not without hope.

Treatment.—Treatment will be directed toward conserving the heart's strength in every way possible and in prolonging the time of rest until danger from rupture of the heart, acute dilatation, etc., seems well over. Not a few errors have undoubtedly been made in regarding some of these cases as simply acute gastric upsets and permitting the patient to begin to move about too soon.

Rest in bed should be strictly enforced. The patient should have his choice as to position in bed, sitting up or lying down. Exceptionally he may have to be allowed to spend his days and nights in the chair. Diet should be light and food producing gas should be avoided. Enemata or cathartics should be given to induce easy stools and to avoid straining.

Morphin may be necessary for the pain. At times this has to be repeated and in fairly large doses. Heat to the chest or sinapism are advised by some. Their value is doubtful.

With the feeble, often irregular, heart digitalis should be given. So far as I know no case of rupture of the heart or other untoward accident due to the use of this drug in this condition has been reported. It strengthens the heart's action and presumably improves coronary circulation. And it is on the establishment of an efficient collateral circulation that any hope of recovery rests. Stimulants, such as camphorated oil, caffein or strychnia, also have a place and should be employed when heart failure seems threatening.

Thrombosis of the coronary artery with resulting infarction will soon have a place in medicine as a definite clinical entity. Already enough is known about it so that it may generally be recognized at the bedside and differentiated from the ordinary paroxysmal angina pectoris. Further histories of carefully observed cases with electrocardiographic studies and autopsy control are desirable. A good résumé of our present knowledge, with many helpful references, is found in a recent article by Wearn.

CARDIAC PAIN

In many diseases of the heart, some of them serious, there is no complaint of pain. This is true of most cases of valvular disease as well as of acute and chronic inflammatory and degenerative processes in the myocardium. Other diseases, however, such as pericarditis, diseases in which angina pectoris is a symptom, certain forms of nervous disturbance as irritable heart and paroxysmal tachycardia, may cause marked pain. In some instances, as in syphilis of the aorta, coronary arteries and aortic valves, it is difficult to state how much of the pain has its origin in the heart proper and how much in the aorta. For practical purposes the two conditions, the cardiac and aortic, may well be considered together.

There is still a lack of exact knowledge as to the origin of cardiac pain. Physiologists are not in agreement as to whether it may be caused by irritation of the intracardiac nerves. The prevailing impression has been that the heart muscle is insensitive to pain. The interpretation of the experimental investigation of pain in animals is difficult. Observations made on human hearts exposed by operation or accident, such as the famous cases of Harvey and Ziemssen, show as the result of pressure on the heart and irritation by means that might be expected to cause pain that patients describe the sensation more as a sense of constriction or of anxiety than of pain. Some observers regard the pain as a reflex phenomenon, the irritant going by way of the sympathetics to the cord and then being reflected outward to certain areas (Head's zones) as a pain sensation. Certain clinical and experimental work lends color to the belief that relative ischemia, such as would be produced in the heart muscle by an insufficient blood supply through diseased coronaries, may cause pain. This was referred to earlier in discussing angina pectoris.

Gieser has recently reviewed the literature on this subject.

But whatever the mechanism of the production of cardiac pain the physician has always to solve the problem of the significance of pain in any given case. The symptom must be interpreted as accurately as possible in terms of changed anatomic structure and disordered function before treatment that may be called rational can be employed. In other words, given "pain in the heart" as a striking symptom, what is the diagnosis? This naturally requires a careful inquiry into the history, a thorough physical examination of the body, as a whole, as well as of the cardiovascular system, frequently with the employment of laboratory and instrumental help. Without this complete examination, errors will be frequent and treatment faulty or more empirical than it should be.

It is not the purpose of this article to discuss the details of diagnosis. It may not be out of place, however, to emphasize the fact that "pain in the heart" which is the complaint of many patients may mean a great many other things than true heart disease. These should all be excluded before deciding that cardiac disease really exists.

In the first place, there is a large group of so-called functional or nervous disturbances in which pain or other distress in the cardiac region is described by the patient. These are often called the cardiac neuroses. The psychic element is here to be carefully considered.

Then it must not be forgotten that disease near the heart may cause pain referred to that organ. Pleurisy, pneumonia, or tumors of the lung may be cited as examples. Mediastinal inflammations, tumors or other masses, such as aneurysms, may induce severe pain. A very common cause of thoracic pain that may resemble cardiac pain is vertebral disease such as tuberculosis, neoplasm, osteoarthritis. Tumors of the cord may also have to be considered. And there are many conditions affecting the wall of the chest that must be excluded: inflammation of the skin or subcutaneous tissue, periostitis or osteomyelitis of the ribs or sternum from such causes as typhoid, tuberculosis, staphylococcus infections, and similar conditions in the cartilage; tumors in the skin, the breast, the bones, the pleura; traumatic lesions as fractured ribs; gouty and rheumatic affections of the muscles, fasciæ and joints of the chest wall; intercostal neuritis, herpes zoster. Also, it must not be forgotten that primary conditions still more remote may serve to explain what seems to be cardiac pain. Tabetic pain may be confusing. Gall-stones, ulcer of the stomach or duodenum, irritable colon, diaphragmatic hernia, acute pancreatitis, cardiospasm, or substernal goiter may cause misleading symptoms.

It is only by excluding such diseases that one may reach the conclusion that the "pain in the heart" really has its origin in that organ. And even then one must remember that a patient may have pain due to more than one cause. I have seen a pain from a carcinoma of the breast in a woman who had an aortic disease; a proved combination of duodenal ulcer with its typical pain and angina pectoris; gall-stones and angina; and often the addition of neurotic complaints to those of a real organic lesion of the heart.

Pericarditis.—Pericarditis may be painless or the pain may be obscured by that of an accompanying disease, such as pleurisy, or may be ignored by the patient because of the depressed mental condition due to toxemia. Clean-cut, outspoken pericarditis causes pain of such severity and such definite localization and is attended by such changes of temperature, character of respiration and pulse rate, and by such typical physical signs, that little difficulty is encountered in recognizing the condition, but in some instances the pain is referred not to the precordia but to the back of the shoulder or even, as in angina, to the neck and arm. More confusing still is the abdominal, particularly the epigastric, reference of pain which may produce a condition resembling subphrenic accident, such as perforation of a gastric ulcer, pancreatitis or cholecystitis. And even with the detection of the pericardial friction and the history of the case, a most careful physical examination may be necessary before one decides that the abdominal symptoms are entirely secondary and rules out a primary subdiaphragmatic inflammation.

The treatment of pericarditis will be more fully discussed elsewhere. It will be sufficient to state here that the treatment should depend in a large measure upon the primary disease, such as rheumatism, tonsillitis or focus of infection in some other part of the body. The treatment, therefore, will depend upon discoverable etiologic factors. Rest in bed, cold to the precordial region, use of opiates when necessary, with salicylates or other remedies supposed to have a specific effect on the primary disease would be the treatment indicated.

Acute Myocarditis.—Acute myocarditis, even the so-called septic form with multiple disseminated abscesses, as in pyemic conditions, may be painless. In acute interstitial myocarditis, however, as in diphtheria, the pain is sometimes quite severe. This fact is worth emphasizing for it may be the first hint of the existence of this serious cardiac condition which, as has been shown by numerous studies, is not infrequently the cause of alarming or fatal complications. The pallor of the face, weaker, more irritable or irregular pulse, perhaps nausea and vomiting, may be present, but often it is the striking substernal pain, at times with some radiation to the neck, that gives the warning. The pain may be referred to the epigastrium; it may be slightly aggravated by movement. While antitoxin has lessened the number of such cases, they are still occasionally seen and the importance of their recognition, so that proper rest may be enjoined, is self-evident. Romberg calls attention to a group of postdiphtheric cases in which he found myocardial lesions with paroxysmal, anginalike, precordial pain with vomiting and even collapse, when the patient seemed well on the road to recovery. I have seen a girl of eighteen, convalescent from diphtheria, suffer almost as in angina from walking merely the length of the ward and the serious strain on the heart made evident also by the ashy color, the dyspnea and small, rapid pulse.

While diphtheria affords the best example of this complication, other diseases, such as typhoid fever, scarlatina, and rheumatism may cause a similar myocarditis, and in these diseases the cardiac symptoms, among which pain may be mentioned, should receive attention.

It is hardly necessary to add that the treatment of this type of disease would include excellent and unusual care during convalescence. Prolonged rest may be the only safe procedure. It may be necessary to give small doses of digitalis. Rarely opium is indicated. It goes without saying that the primary disease should be properly treated.

Valvular Heart Disease.—Valvular heart disease is generally regarded as painless, and yet any physician who will look over his records will find numerous histories showing a complaint of pain in valvular disease of the heart. By this is not meant the sense of oppression or constriction which accompanies the muscularly incompetent heart, but a real pain—something that hurts; and a frank angina pectoris is not meant.

That lesions of the aortic valve, particularly sclerotic and syphilitic lesions resulting in aortic regurgitation, are often accompanied by pain is well known. This pain may be clearly of the classical anginal type, but with mitral disease also there may be pain, particularly with mitral stenosis; short, sharp, painful catches or stabbing pains in the region of the heart, sometimes on exertion, sometimes under excitement or even when the patient is quiet. The pain may be fairly severe; it is usually localized, rarely radiating as in typical angina. When this precordial or submammary pain is associated with the complaint of palpitation and nervousness, and when the patient is clearly neurotic and inclined to exaggerate, it is easy to conclude that the pain is of the so-called functional character, and an error may be more readily made because this pain in mitral stenosis may precede any marked signs of cardiac insufficiency and lead to a rather carelesss examination with the overlooking of the mitral lesion. I have learned to examine with special care for mitral stenosis patients who complain of palpitation, nervousness and even slight dyspnea, and who describe at the same time sharp or stabbing pain in the heart region. One should not in these cases decide too soon that one is dealing with a pure neurosis or with a masked Basedow's disease.

Epigastric discomfort, at times amounting to real pain, due to a passively congested liver might lead one to discuss the importance of the recognition of this condition. It is sufficient here, however, merely to call attention to the fact that misinterpretation is easy unless the thought of probable cardiac disease is entertained.

The treatment of pain in valvular disease and the treatment of distress in the epigastrium with a failing heart carry with them well-known lessons as to treatment. Ordinary rest, light diet, laxatives, and the use of digitalis or other appropriate remedies relieve the condition to a great extent. Some cases of mitral stenosis, however, are peculiarly resistant and in spite of cardiac and nerve remedies the pain continues to be annoying.

Paroxysmal Tachycardia.—Paroxysmal tachycardia is in its prolonged and exaggerated forms often accompanied by pain. This cardiac disturbance and its treatment will be discussed elsewhere.

REFERENCES

Angina Pectoris and Acute Coronary Obstruction

Brit. Med. Journ., p. 1, Jan. 13, 1923.

Coffey and Brown. The Surgical Treatment of Angina Pectoris, Arch. Int. Med., xxxi, 200, 1923.

Cutler, Elliott C., and Levine, S. A. Cardiotomy and Valvulotomy for Mitral Stenosis, Bost. Med. & Surg. Journ., June 28, 1923.

Gieser. Reizperzeption und Zentripetale Reizleitung im Herzen-Zentralbl. f. Herz. Krankh., xiii, 1, 17, 1923.

Hay, John. Cardiac Pain, Lancet, 1, 885, 1922.

Herrick, James B. Clinical Features of Sudden Obstruction of the Coronary Arteries, Journ. Am. Med. Ass., lix, 2015, Dec. 7, 1912.

——— Thrombosis of the Coronary Arteries, Journ. Am. Med. Ass., Ibid., lxxii, 387, Feb. 8, 1919.

Herrick and Nuzum. Angina Pectoris, Clinical Experience with Two Hundred Cases, Journ. Am. Med. Ass., lxx, 67, 1918.

Lutembacher. Douleurs de Distension Cardiaque, Presse méd., April 1, 281, 1922.

McWilliam and Webster. Some Applications of Physiology to Medicine, Brit. Med. Journ., 51, Jan. 13, 1923.

Manouelian. Ann. de l'Inst. Pasteur, xxviii, 582, 1914.

Schmidt, R. Med. Klin., viii, x, Jan. 1 and 8, 1922.

Wearn. Am. Journ. Med. Sc., Feb., 1923.

Wenckebach, K. F. Angina Pectoris and the Possibilities of Its Surgical Relief, Brit. Med. Jour., May 10, 1924.

CHAPTER XIII

TREATMENT OF DISORDERS OF THE HEART BEAT

Paul Dudley White

Introduction.—Disorders of the heart beat are abnormalities of function and therefore their treatment actually amounts to symptomatic therapy. The underlying condition of these various disorders should of course be diagnosed in every case, for the recognition and treatment of the etiological factor is of prime importance. Nevertheless, it is worth while summarizing certain procedures which are known to be of value in relieving some of these disorders of the heart beat.

Disorders of the heart beat consist of disturbances of sinus rhythm itself, ordinarily called sinus arhythmia; premature beats, auricular, ventricular or the so-called atrioventricular or nodal; paroxysmal tachycardia of auricular or ventricular origin; auricular flutter; auricular fibrillation; heart block, partial and complete; and pulsus alternans.

SINUS ARHYTHMIA, SINUS BRADYCARDIA, SINUS TACHYCARDIA

Sinus Arhythmia.—All manner of disturbance of sinus rhythm may occur, from a simple irregularity in rate of the pacemaker in the sino-auricular node to a complete sino-auricular heart-block with auricular standstill. The very common condition of respiratory *sinus arhythmia* is now generally well recognized, but occasionally when exaggerated in a nervous individual it may be confusing. Sometimes it may appear as if the sinus arhythmia were not definitely related to respiration, but a simple test of forced respiration will quickly settle the matter. Normally, forced inspiration produces a definite tachycardia in a patient showing sinus arhythmia, while forced expiration will produce a pronounced bradycardia. This arhythmia is of little importance, certainly not to be treated itself. It is the result of nervous instability in a given patient, the balance between the actions of the sympathetic and vagus nerves on the sino-auricular node being a very delicate one in such an individual. The condition, especially when exaggerated, is very common in young people, particularly between the ages of twelve and twenty-five. It tends to be exaggerated under such conditions as convalescence from infectious disease and nervous or physical fatigue of any sort. The condition itself is not a sign of health; it is a sign of active response of the heart to nervous stimuli. It has been considered a sign of a healthy heart, but it is found sometimes in badly damaged hearts and the reason is obvious: heart muscle may be diseased and still the heart responds in a normal or exaggerated manner to nervous stimuli. Since, however, young people have less heart disease than old people and since arhythmia is more common in young people than in old people, so it may be said that sinus arhythmia and normal heart muscle are usually associated. There is no treatment indicated for sinus arhythmia, as such.

Sinus Bradycardia.—Rarely one finds the condition of so-called *sino-auricular block,* which consists of a very marked slowing of the rate of the pacemaker in the sino-auricular node (down to 40 or below per minute) generally without any auriculoventricular block, or a dropping out altogether for one pulse interval of any evidence of auricular stimulus or contraction, or very rarely a complete sino-auricular block consisting of a standstill of the auricles. In this last-named condition the ventricle escapes, idioventricular rhythm at a slow regular rate taking the place of the previous normal rhythm. This condition of sino-auricular block, whether simply pronounced slowing of the whole heart or "dropped beats" of the whole heart or complete auricular standstill, is generally the result of digitalis intoxication. It is apparently chiefly the effect of marked vagal stimulation. In seven and a half years at the Massachusetts General Hospital there have been only 11 cases; in these 11 digitalis was primarily responsible in 6; in 5 patients the block was complete, giving rise to auricular standstill, and in 4 cases there were dropped beats. In 1 case there was a very slow sino-auricular rhythm and in 1 case the sino-auricular rate suddenly halved after exercise. The conduction intervals between auricles and ventricles when present were within normal limits. The condition was transient in all cases, without effect on life.

Treatment.—If digitalis is found to be the cause of the sino-auricular block the remedy is obvious: namely, the omission of the digitalis. It is probably worth while to try the effect of atropin 1/30 gr. by mouth or even subcutaneously as a therapeutic test in a case of "sino-auricular block" but whether or not repeated doses of atropin or belladonna would tend to abolish the block we have little evidence. In the absence of digitalis a search for other factors giving rise to vagal irritability should be instituted and causes, if any are found, eliminated.

Sinus Tachycardia.—Finally, in discussing variation of rate of the sino-auricular pacemaker, we come to the so-called *sino-auricular tachy-*

cardia, which is simply the rapid rate of the normal pacemaker of the heart, as a rule the result of extracardiac conditions.

Treatment.—Treatment of the tachycardia itself is usually ineffective. An attempt to slow the rate by digitalis or other drugs is probably unwise when the rapid rate is the only disturbing cardiac sign. The condition back of the tachycardia is, of course, what demands treatment. Nearly every infectious disease produces sino-auricular tachycardia. Hyperthyroidism is associated with tachycardia; nervousness gives rise to tachycardia in many people; the toxic effect of uremia results often in tachycardia. Heart failure with normal rhythm may produce tachycardia, but in every instance it is the underlying condition and not the tachycardia which must be treated. As a general rule, rest in bed is the essential therapeutic measure for any of these conditions. If the pulse rate rises above 150, and remains at a uniform rate, some other mechanism than sino-auricular tachycardia should be suspected. Under such circumstances either paroxysmal tachycardia or auricular flutter is probably the underlying condition.

PREMATURE CONTRACTIONS

Premature contractions of the heart, so-called "extrasystoles," are most commonly ventricular in origin, the ratio of ventricular premature contractions to auricular premature contractions being 10 to 1 or greater in frequency. The premature contraction, whether auricular or ventricular, is almost certainly the result of abnormal irritability of the myocardium or of the special conducting tissue. It may be simply a temporary toxic effect associated with indigestion, tobacco poisoning, nervous fatigue or excitement. On the other hand, it may be the result of definite change due to myocardial disease as in arteriosclerosis or rheumatism. It may occur rarely, only a few times in the course of days or weeks, or it may come so often that it produces a bigeminal or coupled pulse, every other beat being a premature contraction. In rare cases, there may even be more premature contractions than normal beats of the heart. Of course, the more often the premature contractions occur the more evidence there is of irritation and irritability of the myocardium. In themselves premature contractions are not evidence of heart disease; they are simply evidence of an irritable or irritated heart. There are records of patients who have shown premature contractions for a great many years without any appreciable heart disease. Mackenzie reports the case of a patient, a man of sixty-nine, who came to him complaining of premature contractions of the heart which had occurred at frequent intervals for over fifty years, causing much distress of mind because of the failure properly to recognize the significance of these premature beats. Even at the age of sixty-nine this man was in fairly good health except for the premature contractions.

Treatment.—It is obvious that in the treatment of premature contractions the underlying cause should be ascertained. This is not always possible, but in at least half the cases clues can be found and the omission of some such offending factor as tobacco, overwork or anxiety may result in the disappearance of the premature beats. Sometimes in the case of heart disease attended by premature contractions, rest and digitalis will result in disappearance of heart failure and the premature beats associated with it. On the other hand, digitalis may itself induce premature contractions if given in large doses, particularly to persons with irritable hearts. Such premature contractions may be either auricular or ventricular and are a sign at the height of digitalis intoxication that the drug should be stopped. If the typical bigeminal pulse appears during the administration of a good deal of digitalis it is probable that a high percentage of the lethal dose has been administered.

Thus there is no set rule about the treatment of premature contractions. Sometimes if heart failure is present digitalis will cause the premature contractions to disappear, and sometimes it will produce them. If treatment of the underlying condition or the institution of proper hygiene in a person who has lived unhygienically fails to abolish the premature contractions, it may be worth while to try drug therapy. Bromids in a few cases seem to help, and it has been recently suggested that quinidin sulphate given in doses of 3 to 6 gr. (0.2 to 0.4 gm.) several times a day may clear up the premature contractions. Certainly quinidin sulphate is ineffective in some cases, but in other cases the premature contractions have disappeared under its administration at the same time that there has been an increase in pulse rate. Further work in the use of quinidin sulphate in the treatment of obstinate premature contractions must be done to establish its value.

Summarizing the therapy of premature contractions, it may be said that the underlying factor is to be searched for and treated. The frequent failure to discover and remedy the underlying cause makes it advisable at times to try therapeutic measures such as the administration of bromids or quinidin; but, as Mackenzie has said, not infrequently, regardless of the therapy instituted, premature beats will persist for many years and not incapacitate the individual showing them. Reassurance is very frequently an important measure in the treatment of a person bothered by the palpitation of premature beats. Mackenzie finishes his discussion of the treatment of "extrasystoles" by saying that some people have premature contractions more often when they are leading sedentary lives indoors. Therefore he advises for such people more exercise in the open air.

The separation of premature contractions into auricular, ventricular and nodal has little if any significance in regard to treatment. It is thought that auricular premature contractions, although much more rare, are more significant evidence of cardiac irritability and are apt to precede such conditions as paroxysmal tachycardia or auricular fibrillation or flutter. The atrioventricular or nodal extrasystoles are very rare and consist essentially of an escape of the ventricular pacemaker due to excessive irritability of the atrioventricular node. Digitalis may be responsible for some of these cases.

PAROXYSMAL TACHYCARDIA

Paroxysmal tachycardia consists in sudden acceleration of the rate of the heart, lasting seconds, minutes or hours, and stopping abruptly. The condition is due to the inception of abnormal rhythm by some irritable focus in the auricular or ventricular myocardium. The rate of this abnormal pacemaker ranges usually between 120 and 200 and a very frequent rate is in the vicinity of 160 per minute. Auricular paroxysmal tachycardia, that is, the abnormal rhythm resulting from an irritable focus in the auricle, is much more common than the ventricular type of paroxysmal tachycardia, the ratio at the Massachusetts General Hospital in the last eight years being about 25 to 1. Why this is so is not known, since ventricular premature beats are so much more common than auricular premature beats, and it is ordinarily considered that paroxysmal tachycardia consists of a rapid repetition of premature contractions. Certainly further investigation of the fundamental mechanism of premature contractions and paroxysmal tachycardia is essential to clear up this point. Clinically, the underlying causes of paroxysmal tachycardia are just the same as those for premature beats. Paroxysmal tachycardia may occur in organic heart disease and is not infrequent in cases of thyroid heart disease, arteriosclerotic heart disease or rheumatic heart disease, but it is much more commonly found in the absence of organic heart disease and in the presence of irritability or irritation of the myocardium. Again, toxic influences such as digitalis, tobacco, indigestion and fatigue may produce paroxysmal tachycardia.

General Treatment.—The treatment of paroxysmal tachycardia consists in the discovery and omission of the exciting cause. Here, as in the case of premature contractions, it is not infrequently impossible to ascertain the etiology. Then symptomatic therapy must be instituted, and there are almost as many methods recommended for the immediate treatment of paroxysms of tachycardia as there are patients who have paroxysmal tachycardia. A good many victims have discovered and utilize their own methods for stopping attacks, and nearly every textbook in discussing paroxysmal tachycardia gives a list of methods which have been found of value in stopping the attacks in various individuals. Most of these methods depend on the reflex stimulation of the vagus nerve and probably the cases successfully treated under almost any method are those in which the abnormal pacemaker is more under the influence of the vagus nerve than it is in the cases which fail to be influenced.

Some of the procedures which have been used with success are, in brief, as follows: vagal pressure on either side of the neck, consisting of firm digital compression of the carotid artery midway in the neck (compression of the carotid artery to the point of occlusion of the vessel will produce stimulation of the vagus nerve which lies in the carotid sheath under the artery), and pressure of the eyeball on either side with the eye closed, up to the point of pain. Both these measures are of more value than all the other procedures put together and they certainly should be tried in every case of paroxysmal tachycardia. Pressure on the vagus nerve, if successful, usually is effective in less than a minute, sometimes in a very few seconds; similarly with regard to ocular pressure. The vagal pressure is a direct stimulation of the vagus nerve while the ocular pressure resulting in the oculocardiac reflex is a result of indirect reflex stimulation. These measures are effective in probably one-third of the cases of paroxysmal tachycardia. Some patients can be taught to compress their own vagus nerve at the onset of an attack of paroxysmal tachycardia and so control the condition themselves without the need of calling a physician. Other procedures, mostly affecting the heart through vagal reflex action, are the induction of vomiting, repeated swallowing or the swallowing of a considerable amount of cold water, an attempt at forced expiration with the glottis closed (the so-called Valsalva experiment), holding the breath as long as possible, leaning over backwards with the neck extended (resulting in stretching the tissues of the neck), firm compression of the abdomen or chest by a swathe, ice-bag to the epigastrium or precordia, and finally leaning forward with the head very low. All these measures have resulted in stopping paroxysms of tachycardia in different individuals. Sometimes the same procedure will be effective repeatedly in a given person and sometimes one of these procedures will be effective in the treatment of paroxysmal tachycardia in several different individuals. It is doubtful whether the type of paroxysmal tachycardia, that is, auricular or ventricular, is of importance in relation to the therapeutic measure to be used.

Drugs.—A word should be said about the drug therapy of paroxysmal tachycardia. Digitalis has been given intravenously, subcutaneously and by mouth to patients with paroxysmal tachycardia. Strophanthin has also been used, morphin has its advocates, and now quinidin sulphate is being recommended. Certainly in some patients with paroxysmal tachycardia the attacks have stopped very soon after the administration of some of these drugs. It must always be borne in mind, however, that paroxysmal tachycardia stops spontaneously and that an attack may stop spontaneously within a short time after the administration of some drug. If, however, the patient has paroxysmal tachycardia, usually of a fairly definite duration and repeated, and the administration of some such drug as digitalis, morphin or quinidin sulphate results in a marked shortening of the attack, it is reasonable to suppose that the drug has had an effect. One must view all remedies, however, very critically. At the present time it is believed that quinidin sulphate in some cases does stop attacks of paroxysmal tachycardia. The dosage should be 6 gr. (0.4 gm.) at two-hour intervals in a person who is not susceptible to quinidin intoxication. (The test for susceptibility consists of administration of 3 gr. (0.2 gm.) with observation as to the appearance of headache, nausea or tinnitus for the next few hours.) At the Massachusetts General Hospital we have had success with quinidin in two or three cases and in as many more failure. That has been the general experience elsewhere, so that the present status of quinidin therapy in paroxysmal tachycardia is uncertain. Further investigation should be carried on. One more point should be mentioned regarding the use of quinidin sulphate, and that is that the daily rationing of from 3 to 12 gr. (0.2 to 0.8 gm.) may be of some value in the prevention of paroxysms of tachycardia.

Finally atrioventricular paroxysmal tachycardia, or nodal paroxysmal tachycardia as it is sometimes called, is very rare. The treatment would be the same as for the other types.

AURICULAR FLUTTER

Auricular flutter and auricular fibrillation are closely allied conditions, but since there is some clinical importance in differentiating them they will be discussed here separately. Auricular flutter consists of a very rapid regular contraction of the auricle at rates between 200 and 400 with the ventricular response usually at one-half the auricular rate, that is with 2 to 1 heart-block accompanying the auricular flutter. The reason for the heart-block is obvious. The auriculoventricular conduction tissue is unable to transmit, as a general rule, every auricular impulse. Rarely does one see a patient with auricular flutter without block, in which every stimulus descends to the ventricle, even at rates close to 300. The important work of Thomas Lewis during the past two years in analyzing the mechanism of auricular flutter and auricular fibrillation has demonstrated that auricular flutter is due to a circus movement in the right auricle. This circus movement consists of a contraction wave traveling rapidly along a band or bands of muscle around the great veins. Apparently a certain degree of irritability of the auricular muscle is necessary before this circus movement is initiated. This degree of irritability is dependent, as a rule, on definite cardiac disease and so auricular flutter is seen chiefly in two conditions: arteriosclerotic heart disease and rheumatic heart disease. Rarely it may result from other conditions such as temporary toxemia. Hence auricular flutter is of more clinical significance, in other words, a more important sign of heart disturbance, than is paroxysmal tachycardia, and its mechanism is quite different so far as we know at present.

Treatment.—Some years ago Ritchie and others described what has been a classical method of treating flutter. Upon the discovery of the condition by electrocardiogram or jugular pulse tracing, for it cannot be proved by other methods of examination, the heart is saturated with digitalis. The first effect from the digitalis is the increase in the degree of heart-block so that 3 and 4 and even 5 and 6 to 1 auriculoventricular block results. Sometimes this block occurs regularly so that the ventricular rate will suddenly drop from 150 to 75 for example, and sometimes the block will be irregular in grade so that a high degree of arhythmia results from the rapid succession of grades of block varying from 2 to 1 up to 6 to 1. By careful analysis of the pulse even these irregular grades of block can be analyzed correctly through the law of dominant rhythm. If the digitalis is pushed to a still further degree, perhaps up to the point of gastro-intestinal discomfort, auricular fibrillation is instituted in a considerable percentage of the cases. As soon as auricular fibrillation appears the digitalis is discontinued and in certain cases normal rhythm quickly results. However, in a certain number of patients this classical method of treatment does not have the results expected and on stopping the digitalis the auricular fibrillation may persist or flutter recur. In spite of the failure in many cases of auricular flutter to yield properly to digitalis, digitalization is a method still in vogue and often useful, for even though the auricular flutter is not abolished, the ventricular rate is controlled by the production of block. With a regular ventricular rate of 75 to the minute proper circulation can be maintained indefinitely even though the auricles are continuing their contractions at the rate of 300 a minute. Maintenance of digitalization would be necessary to cause this effect to persist. The procedure for maintenance of digitalization would be the same as that to be described shortly under auricular fibrillation. Moreover, even if auricular fibrillation is produced by the digitalization of auricular flutter, it may be controlled, so far as ventricular rate is concerned, by the maintenance of the digitalization.

Since the introduction of quinidin sulphate, however, a new method for the treatment of flutter has been instituted. Sufficient work has not yet been done to be sure of the wisdom of giving quinidin sulphate to most cases of auricular flutter. In some cases the drug has been effective in abolishing the flutter. In other cases, the flutter has persisted in spite of the quinidin and has even been made apparently more obstinate in its reaction to digitalis, which was tried later. The treatment of flutter by quinidin is similar to that of fibrillation by quinidin.

The principles of the treatment of auricular flutter are two in number: first, attempt to abolish auricular flutter altogether with a restoration to normal rhythm, whether by the use of digitalis or quinidin; second, if the first procedure is impossible, control the ventricular rate in auricular flutter by proper digitalization. It must be borne in mind, however, in conclusion, that auricular flutter is often paroxysmal and may stop spontaneously. If the attacks are very brief, as sometimes happens, neither digitalis nor quinidin would have time to get in an effect before the attack is over. It should be said, however, that paroxysms of auricular flutter, just as paroxysms of auricular fibrillation, may be decreased in frequency and even abolished altogether for a considerable length of time by rations of quinidin sulphate. Amounts of quinidin sulphate either in tablet or capsule form, 3 gr. (0.2 gm.) at a dose, and from 3 gr. to 12 gr. (0.2 to 0.8 gm.) a day, are effective in some cases in the prevention of paroxysms of auricular flutter. Therefore, in a patient who shows paroxysms of auricular flutter it is worth while to try the effect of quinidin sulphate as a prophylactic. If the attack of auricular flutter has become well established either digitalis or quinidin sulphate may be tried. At the present moment it is probably wiser to use digitalis by and large. It is desirable that each case should be tried individually, for some patients will react to quinidin better than others and it may actually be injurious to some if quinidin therapy is persisted in after early failure to react.

AURICULAR FIBRILLATION

Auricular fibrillation is a very common disorder of the heart beat, very much more common than auricular flutter clinically, probably seen twenty times more frequently. As a matter of fact, as the investigations of Thomas Lewis and his collaborators have shown, the mechanism behind the two conditions is the same, but auricular fibrillation is a higher grade and more prominent type of the disturbed mechanism than is auricular flutter. In the case of auricular fibrillation, the so-called circus movement which travels very rapidly around the great veins of the right auricle reaches such a speed that it no longer travels uniformly but becomes very irregular in its course and rate. Auricular rates of 500 or more are not infrequent in auricular fibrillation. The ventricles respond irregularly and rapidly to the circus movement of auricular fibrillation so that clinically there is a sharp distinction between auricular flutter and auricular fibrillation. There are occasionally border-line cases in which by electrocardiogram auricular activity is seen to be not quite regular and yet closely resembling flutter. This condition is sometimes called impure flutter or coarse fibrillation. Such border-line cases would be expected to occur. Their treatment is similar to that for auricular fibrillation.

Absolute arhythmia, which is the old term given to auricular fibrillation, may be paroxysmal or permanent. It is generally a sign of serious or important heart disease. The most common etiological causes for heart disease associated with auricular fibrillation are coronary arteriosclerosis (abbreviated to *cardiosclerosis*), rheumatic heart disease and thyroid heart disease. Although auricular fibrillation is almost always a sign of extensive heart disease it may be borne for many years, and cases are on record who have shown absolute irregularity of the pulse for twenty or thirty or more years.

The chief reason, clinically, why auricular fibrillation is a burden to the circulation is not because the auricle contracts ineffectually but because the ventricular rate is high. The ventricular contractions are so rapid and irregular that the circulation is badly disturbed.

Digitalis Therapy of Auricular Fibrillation.—There are two methods of treating auricular fibrillation. The first, which is in general the one widely accepted and which should be continued in most cases, is by the use of digitalis. By the digitalization of a patient, that is, by the saturation with digitalis, there results a depression of the conducting mechanism of the heart. The ventricles can no longer respond to so many rapid irregular stimuli from the auricle, and the partial block which is already present is increased. When the increase of block has reached such a point that the ventricular rate is reduced from its original 120 to 160 down to close to a normal figure, 70 to 80, the circulation improves tremendously. Improvement of the circulation is due almost entirely to the more effectual contractions of the heart. When the heart is contracting irregularly at the rate of 160, the force of many of the beats is so slight that the pulsations do not reach the periphery of the body. Hence the contractions of the heart are wasted and much needless work is done. Particularly is this injurious to the heart and circulation if the myocardium is diseased, as it is in most of the patients who show auricular fibrillation. The heart muscle becomes tired and a vicious circle is instituted. Congestive failure results, and unless the ventricular rate is cut down the patient may die of heart failure. By the simple reduction of the heart rate from 150 to 75 a minute by the use of digitalis in auricular fibrillation, 75 beats a minute are saved. The work of the heart is reduced one-half. In the course of twenty-four hours 108,000 contractions of the heart would take place instead of 216,000; there would be a saving to the heart of 108,000 beats a day. This must mean tremendous rest for the heart, and it is that rest with improvement in the force of contractions that restores the circulation so rapidly and dramatically in patients with auricular fibrillation and congestive failure, who have been previously improperly treated. To be sure, digitalis also acts by increasing the degree of systolic shortening of the heart muscle, and so, often when the pulse is normal and slow, digitalis has a beneficial effect in cardiac congestive failure. This effect would be produced in auricular fibrillation as well as in normal rhythm, but without doubt the most important benefit from digitalis in auricular fibrillation is through the rapid reduction of the ventricular rate. And also without doubt the great reputation that digitalis has obtained throughout the world results from its toxic effect on the conducting tissue and its production of heart-block in auricular fibrillation and not from its stimulating effect on the myocardium. It is surprising to find how few doctors recognize the importance of this point although Withering called attention to it over a century ago. The principle of digitalization has only recently become understood by any considerable percentage of practicing physicians.

The principles of digitalis therapy in auricular fibrillation are as follows: first, saturation of the patient with the drug; second, maintenance of saturation as long as necessary. Saturation of the patient with the drug is dependent on several conditions. First, a drug of proper potency is necessary. Fortunately at present most digitalis preparations on the market, if fresh, have been standardized and are within reasonable range of normal strength. If, however, digitalis is used which has been on the shelves of a country pharmacy for some years there may be very little value left in the drug. If in doubt, it is wise either to have preparations of digitalis which are to be used standardized and fresh, or else to use trade preparations which are known to be well standardized. The second point in regard to saturation of a patient with digitalis concerns the time during which saturation can be effected. It is no longer necessary to wait one, two or three weeks before the patient is under the control of the drug. We now know that we can saturate almost any patient in the course of hours, or a day or two at the most. Rarely is there so much urgency that saturation must be effected within a few hours. If such a case is being treated the large Eggleston dosage is advisable. According to the Eggleston dosage the amount of digitalis necessary to saturate is figured up according to body weight; from 0.1 to 0.15 gm. of a potent leaf for 10 pounds of body weight is the standard. After calculation of the total amount necessary for saturation by this method, from one-third to one-half the total amount is given at once; in six hours one-sixth to one-fourth more is given; in six more hours, one-eighth of the total dose, and so on until the patient's pulse rate has been reduced close to normal limits or until other toxic symptoms have appeared, such as nausea and vomiting or a bigeminal pulse. The majority of patients do not need such rapid saturation, and in the course of two or three days enough digitalis may be given to control the ventricular rate. For example, 3 gr. (0.2 gm.) three or four times a day for two or three days will generally produce a sufficient effect quickly enough without running quite so much risk of the production of nausea and vomiting in a sensitive person. It is important however to judge the amount of digitalis by the weight of the individual, and we have Eggleston to thank for pointing out the importance of this point. Occasionally, as a therapeutic test in a patient who may not be under close control but who is to be seen in one week, say in a dispensary cardiac clinic, 1½ gr. (0.1 gm.) three or four times a day may be prescribed. In the course of a week such a patient will generally be saturated and when he returns to the clinic one can decide whether or not there has been benefit from this procedure of a rather slow saturation.

In the discussion of the digitalis treatment of fibrillation I have spoken of grams of the leaf. Of course cubic centimeters of the tincture of a good digitalis leaf may be used instead, at ten times the figure computed for the dry leaf. For the infusion made from the tincture the amount must be still further multiplied by ten. An important point should be brought out here regarding the use of the tincture. Very often I have seen the tincture prescribed in drops when minims were intended. We know that as a rule it takes 2 to 3 drops of the tincture of digitalis to make 1 minim. Therefore, unless this point is recognized and sufficient dosage is ordered to make up the minims planned, or unless a minim glass is available, the use of the tincture is uncertain. The measurement of drops is rather cumbersome and the carrying about of the tincture of digitalis is not so simple as the carrying of the dry leaf in pill form, each pill comprising 1.5 gr. (0.1 gm.), a very convenient amount for calculation and administration. It is possible that the tincture of digitalis in some patients may be better absorbed than the dried leaf, but for some years, at the Massachusetts General Hospital, the dried leaf in pill form has been used without any appreciable difficulty concerning absorption.

Digitalis may be administered rectally in the same dosage as by mouth if there is vomiting not the result of digitalis.

Another point may be brought up concerning the use of digitalis in auricular fibrillation, and that is the relative value of the proprietary preparations and a simple leaf in pill form. If the leaf is well standardized there is little need to call on any of the expensive proprietary preparations. Eventually, when we have learned what the important active principles of the digitalis leaf are and have a preparation containing simply those principles, we can dispense with the whole leaf. Until that time I think the simplest procedure in the use of digitalis is the use of the leaf in pill form.

Rarely is it necessary to administer digitalis intravenously or intramuscularly, or subcutaneously. Once in a while in an urgent case such a therapeutic measure is advisable. Under such circumstances a preparation of digitalis leaf at the dosage of 1.5 gr. (0.1 gm.) in solution in an ampule may be administered intravenously at intervals far enough apart so that intoxication will not occur unexpectedly, symptoms and signs of intoxication to be observed before the next dose is due. Probably digitalis may in this way be given every hour or two with care. It is, however, not more than once in a hundred times that I have found it necessary to use digitalis intravenously. Under such circumstances strophanthin may also have a place, although it probably does not act in equivalent strength more quickly than digitalis. The dosage of 1/60 gr. (1 mg.) is rather dangerous for ordinary use; probably ½ mg. is as much as should be given at a time. In the use of any intravenous preparations or in the administration of large amounts of digitalis orally, care should be taken that the patient has not already been taking digitalis in appreciable amount.

The second main principle in digitalis therapy is the maintenance of digitalization when it is indicated, and in the treatment of auricular fibrillation, when it has been decided not to try the effect of quinidin sulphate, such a procedure is almost invariably necessary. After saturation with digitalis, when the ventricular rate has been reduced to about normal, this rate may be maintained for days, weeks, months or years by proper rationing with digitalis. As a rule, it has been found that if as much digitalis is given every day as is excreted, this equilibrium can be maintained. Pardee and others have found that about 0.1 gm. of digitalis is excreted by the average adult every day. Therefore, the administration of this amount 1½ gr. (0.1 gm.) every day for years will maintain a good pulse rate for this length of time. Occasionally, individual patients will find that this amount is too much, that is, they will become toxic when using it. If so, they may occasionally omit the digitalis for a day, or take it every other day. Also, occasionally patients will be found who do not get enough digitalis in this way to maintain saturation. Such patients will need an occasional extra pill, sometimes as much as two pills a day. The various other members of the so-called digitalis group, strophanthus, squill, apocynum and convallaria, have been shown to be inferior to digitalis in cardiac therapy. Therefore these preparations need not concern us further.

Too much emphasis cannot be laid on the two chief principles of digitalis therapy: the proper digitalization of a patient with auricular fibrillation and the maintenance of that digitalization.

Quinidin Therapy of Auricular Fibrillation.—Now we come to the other and more recent method for the treatment of auricular fibrillation, namely, by the administration of quinidin sulphate in the attempt to abolish this abnormal rhythm. For many years certain physicians on the Continent, particularly, had found that quinin combined with digitalis, or given alone, seemed to act beneficially at times on the heart, but no analysis was ever made of this beneficial effect. In 1914, Wenckebach reported that in 2 cases of auricular fibrillation he had succeeded in restoring normal rhythm by quinidin. The first case which called his attention to the possibility of the use of the drug for this purpose was a Dutchman who had spent some time in the tropics, where he had acquired malaria. In addition to malaria he suffered from paroxysms of auricular fibrillation. He discovered himself that when he took quinin, given for his malaria, he secured relief from the paroxysms of auricular fibrillation. This discovery he reported to Wenckebach and Wenckebach was discerning enough to appreciate its significance. Following this finding various other alkaloids of cinchona bark were tried to determine their effect on auricular fibrillation. It was discovered in this way that quinidin sulphate acted much better than quinin or any of the other alkaloids from this bark in abolishing auricular fibrillation, and reports of clinical investigation with quinidin have become more and more frequent from all over the world. From one-half to two-thirds of all the patients with absolute irregularity of the pulse have had a restoration to normal rhythm, but in a certain considerable percentage of these cases fibrillation has recurred.

At the same time that quinidin sulphate was being introduced clinically Thomas Lewis was experimenting on the mechanism of auricular fibrillation and auricular flutter. He has shown in the past two or three years of work on animals and man that the mechanisms of auricular flutter and fibrillation are closely allied and that auricular fibrillation does not consist, as previously believed, in the simultaneous and dissociated activity of many different parts of the auricular musculature. Instead he has shown that auricular fibrillation is the result of a very rapid circus movement of a wave of contraction around the great veins. As has already been stated in the discussion of auricular flutter, the circus movement of auricular fibrillation travels at a faster speed than that of flutter. The action of quinidin is to put an end to this circus movement with a return of function of the normal pacemaker of the heart, namely, the sino-auricular node.

One of the aims of investigation during the past year or two has been to determine what type of case of auricular fibrillation is best treated with quinidin. Quinidin therapy, besides failing in a certain percentage of patients, also is not without danger, the chief danger consisting of the production of embolism upon the reëstablishment of normal rhythm. In auricular fibrillation there is a tendency to stagnation in the auricles; this stagnation sometimes results in thrombosis, particularly in the auricular appendages. Upon the resumption of a regular contraction of the auricles some of this thrombus may be pumped out into the circulation and result in embolism, which may kill the patient. Therefore, in the selection of cases of auricular fibrillation for quinidin therapy care should be exercised to pick out those patients in whom there is little likelihood of auricular thrombus. Such patients are those that have had auricular fibrillation for a relatively short time, that is for weeks or months, or at the most a year or two, those cases of fibrillation which have not had congestive failure of any appreciable degree past or present (for in cases with failure the circulation, being more sluggish, is apt to favor thrombus formation in the auricles), and those cases with auricular fibrillation and well-marked mitral stenosis (for in mitral stenosis there is apt to be auricular thrombosis). Fortunately the very cases in whom there is the least danger of embolism are the cases that respond best to quinidin sulphate in the restoration of normal rhythm and in the maintenance of this normal rhythm. About one-third of all the cases of auricular fibrillation are favorably influenced by quinidin and that third consists chiefly of patients with paroxysmal or recent auricular fibrillation without failure or mitral stenosis. It is of little avail to restore normal rhythm if within a day or two or a week or two there is a relapse to auricular fibrillation; the very patient where there is the most danger of embolism and who responds to the drug usually with more difficulty in the first place is more apt to relapse quickly.

There is then a definite place for quinidin therapy in the treatment of heart disease, but this treatment must be limited to the third of the group of patients with auricular fibrillation described above, possibly also to some cases with auricular flutter and paroxysmal tachycardia, and perhaps to some with premature beats.

Another danger with quinidin therapy that is easily averted and very unlikely to occur is due to individual idiosyncrasy to the drug. Rarely one meets patients who are hypersensitive to quinin; such patients are equally hypersensitive to quinidin. All patients should be tried out with small doses of the drug before giving the larger doses necessary to restore normal rhythm. The toxic effects of quinidin are exactly those of quinin, ringing in the ears, nausea, headache, and, if extreme, respiratory paralysis.

A practical method for the administration of quinidin sulphate is as follows: On the first day test doses of 3 gr. (0.2 gm.) each are given at two or three-hour intervals, say at 2 P. M. and 4 P. M. The drug may be given either in capsule or tablet form. I have had equally good results with both methods. These initial small doses are given to test the sensitiveness of the individual to the drug. The following day, provided there has been no toxic effect from the quinidin, the administration begins in larger dosage: 6 gr. (0.4 gm.) are given every two hours for five doses, say at 8 and 10 A. M., 12 M., and 2 and 4 P. M. On each occasion, before the next dose is due the patient should be examined to determine whether or not there are toxic symptoms, or whether normal rhythm has been restored. If either condition is found, the drug should be discontinued for the rest of the day at any rate. These large doses of quinidin sulphate, that is, 2 gm. or 30 gr. a day, may be continued for several days even up to a week or ten days.[1] Sometimes four and five to six or eight days pass by before the pulse returns to normal. Most of the successful cases, however, are restored to normal within the first two or three days, and some of my cases at the Massachusetts General Hospital have returned to normal after one or two or three doses on the first day.

---

[1] In a few patients, after failure to control fibrillation, a second course of quinidin, with doses as high as 60 grains a day for a few days, has geen given.

Finally, comes the question of the rationing of cases with quinidin after restoration of normal rhythm to prevent the occurrence of paroxysmal or permanent fibrillation. There is no doubt that quinidin sulphate in daily rations of 3, 6, 9 or 12 gr. (0.2 to 0.8 gm.) is effective in abolishing or diminishing in number, and shortening in duration, paroxysms of auricular fibrillation. There is, however, doubt as to whether it is wise to ration every case after restoration of normal rhythm. I believe one must experiment in individual cases. Some patients of mine have had normal rhythm now for a year or more and have not needed to take any quinidin since their auricular fibrillation was abolished. Other patients have relapsed and on the restoration to normal rhythm a second time have done better on daily rations. There does not seem to be any injurious effect from long continued use of the quinidin daily in small doses, regarded as rations. The great subjective relief from the palpitation occasioned in some patients by auricular fibrillation is an important reason in itself for the use of quinidin.

HEART-BLOCK

Heart-block may be one of three types: sino-auricular, which has already been discussed under sinus arhythmia; auriculoventricular, which will be discussed in the present section; and intraventricular, which will also be discussed.

Auriculoventricular heart-block consists of retardation of the conduction of the stimulus for the heart beat from auricles to ventricles. It may be of any grade from the slightest delay in conduction, as shown electrocardiographically by a long *P-R* interval or on the jugular pulse tracing by a long *a-c* interval, up to complete heart-block with a slow ventricular rate of 25 or 30. The cause of the high-grade auriculoventricular heart-block is almost invariably arteriosclerosis of the coronaries; occasionally syphilis or rheumatic heart disease will be the factor.

Intraventricular block of all grades is of more serious significance than auriculoventricular heart-block. For its discovery the electrocardiogram is essential. It also is almost invariably produced by arteriosclerosis; rarely syphilis or rheumatism may be a factor.

The lesser grades of auriculoventricular heart-block, especially the slightest grades, are most frequently produced in the clinic at large by digitalis, and in the study of the cause of heart-block in any given case great attention must be paid to the question of the quantity of digitalis administered and to the susceptibility of the individual patient to the drug. In this the study of the *T* wave of the electrocardiogram helps us a good deal, for the *T* wave is influenced by digitalis even sooner than the *P-R* interval, which is the conduction time between auricles and ventricles. If the *T* wave is flattened or inverted we can feel fairly sure, in a given case, that the digitalis administered is responsible for some at least of the block present. Very rarely can digitalis produce temporary intraventricular block.

With a high-grade auriculoventricular block, partial or complete, there tend to be in some patients spells of faintness or even syncope, and, if long continued, epileptiform convulsions. The association of such spells with heart-block is called the Stokes-Adams syndrome. The spell is nothing more nor less than a condition of cerebral anemia resulting from the temporary asystole of the heart due to the high-grade block.

Treatment.—The treatment of heart-block is of two types: first, etiological, and second, symptomatic. If we know the cause we should so far as possible try to remove it. For example, if we are quite sure that digitalis has produced the block we should omit the digitalis if there is not a clear indication for continuing the drug in the presence of congestive failure. If syphilis is found to be the cause of the block, antiluetic treatment is indicated and occasionally cases have been seen and sometimes reported showing the striking cure of even high-grade heart-block under such conditions. But since arteriosclerosis is the cause in most patients who show well-marked auriculoventricular block and intraventricular block very little can be accomplished in the treatment of the cause. Until we can cure arteriosclerosis most cases of high grade heart-block, whether auriculoventricular or intraventricular, will resist all our attempts.

Symptomatically heart-block can be helped occasionally, but very rarely does heart-block produce symptoms. There may be coincident angina pectoris or congestive failure, but such conditions are not the result of the heart-block and so do not enter into the present discussion. Rarely, however, as I have already said, high-grade auriculoventricular block may be associated with the Stokes-Adams syndrome, and in the treatment of a given attack of Stokes-Adams syndrome such drugs as depress vagal action or stimulate sympathetic action are indicated; for example, adrenalin, atropin sulphate, amyl nitrite inhalations, ammonia, caffein and other stimulants. There is no one single therapeutic measure which is always effective under such conditions and it is worth while in emergencies to try one after the other. Since the syndrome is apparently the result frequently of vagal inhibition of conduction or impaired conduction, the first remedies ordinarily tried would consist of drugs to paralyze the vagus. Often the attacks are so transient, however, that there is no time to give any remedy before the attack is over. To avert attacks various therapeutic measures have been devised. The occasional use of nitrites or of atropin or belladonna is unsuccessful in most cases. Thyroid extract in daily doses (gr. i to vi) seems to help in some cases and tends to maintain a high ventricular rate and so improve the circulation in patients with high-grade block, either partial or complete. Barium chlorid has also been used to stimulate ventricular activity and so to prevent the Stokes-Adams syndrome.

PULSUS ALTERNANS

Pulsus alternans is one of the most important and serious of all disorders of the heart beat. It is a sign of an exhausted myocardium. It consists of regular alternation in force of the heart beats and can be detected, as a rule, only by examination of the arterial pulse, best either at the elbow by the use of the sphygmomanometer or at the wrist by the sphygmograph. Rarely can alternation be detected easily by the fingers palpating the radial pulse. If it is found in this way it is of high grade. The simplest method is that with the use of the sphygmomanometer. In such an examination it is discovered that alternate beats which occur perfectly regularly in time have a pressure from 2 to 3 up to 10, 20 or even 30 mm. below that of the preceding beats; that is, the pulse rate can be halved when the pressure in the cuff is raised to a certain level. The mechanism of the production of pulsus alternans consists probably in partial asystole of the heart muscle, that is, due to improper circulation or to intoxication or fatigue the refractory period of certain heart muscle fibers is prolonged to such a degree that they cannot contract with every stimulus. Therefore, during alternate heart beats some of the muscle fibers are lying idle recovering sufficient reserve to contract with every other stimulus.

Pulsus alternans is found particularly in patients who show hypertension and myocardial weakness. Hypertensive and arteriosclerotic heart disease are the conditions most often showing pulsus alternans. Occasionally any type of heart disease, however, will show the condition provided the heart muscle is weak enough. The presence of absolute arhythmia, in auricular fibrillation, prevents one from discovering the condition of the heart that would produce pulsus alternans, for, of course, pulsus alternans cannot be diagnosed when auricular fibrillation is present. Premature contractions, however, are often very helpful in revealing the presence of pulsus alternans, for following premature contractions there is a tendency for pulsus alternans to develop for a few beats in the borderline cases. Even this degree of alternation of the pulse is important and shows myocardial exhaustion.

Generally speaking, the finding of pulsus alternans entails a poor prognosis and most patients showing it have shortened lives. Usually the patient dies within a year or two of the discovery of the alternation.

Rarely, with great care, a patient may live for even five or six years after the discovery of the condition.

There is one occasion when pulsus alternans is not of serious omen and that is during paroxysmal tachycardia, when after hours or days the heart muscle becomes fatigued and shows temporary alternation of the pulse.

Treatment.—The treatment of pulsus alternans consists simply in the treatment of the myocardial exhaustion. Rest is the first essential and usually absolute rest in bed is indicated for days, weeks or months as long as the myocardial exhaustion persists. Digitalis is indicated generally, and should be given at least a therapeutic trial in most cases. It used to be believed that digitalis was contra-indicated in pulsus alternans; this idea is no longer held by those who have investigated the condition. Jean Heitz and some of his associates have found that some of the patients who show pulsus alternans show also nitrogen retention in nephritis with hypertension. It is quite possible that poisoning resulting from renal insufficiency is a factor in a number of cases and the treatment of nephritis as well as the treatment of the cardiac weakness should be carried on.

CHAPTER XIV

CARDIAC NEUROSES

James B. Herrick

There are numerous patients in whom cardiac symptoms appear when there is no evidence of organic change and where the condition seems to be due to a nervous disturbance. These cases are loosely spoken of as "cardiac neuroses." Some are instances of what might be called a cardiophobia, a fear of heart disease. This centering of the thought of the patient on the heart may have its origin in some case of heart disease in the family, there being an element of imitation, or may have been aroused when some doctor asked questions pointing to heart disease or mentioned a rapid pulse or a skipped beat. A neurotic individual may under these circumstances easily imagine symptoms, or unconsciously exaggerate slight disturbances and become the victim of a cardiac neurosis. The psychic element here plays the leading part. Overwork, worry, anxiety, anemia, alimentary disturbances may all be contributing factors.

The discussion of the treatment of such a condition would be the discussion of the treatment of the nervous state rather than the cardiac. It involves reassurance and encouragement, attention to diet and exercise, relief from overwork, and all that goes with the treatment of psychasthenia or neurasthenia. In its exaggerated form the cardiac neurosis is seen in the so-called irritable heart. Under this head more will be said as to symptoms and management.

THE IRRITABLE HEART

In spite of the study of many thousands of cases in the armies of all the belligerents in the late World War, the results of which studies are embodied in hundreds of articles in many languages, there has not come forth any simple, satisfying explanation of the peculiar syndrome known as "irritable heart." In fact there is no agreement as to the name that should be attached to this group of symptoms. DaCosta who gave the first accurate description of the condition called it the irritable heart of soldiers, which term has sometimes been abbreviated to "soldier's heart." But because the same condition is seen in civil practice the term "irritable heart" seems preferable. Other names are cardiac neurosis, effort syndrome, neurocirculatory asthenia, cardiac instability, vagasthenia. Mackenzie at one time called it the "X" disease, a name still not unappropriate as admitting frankly that its real nature is unknown.

The patients may be children or adults. Generally they are young adults. In civil life the two sexes are fairly equally represented. Under physical or emotional strain that would be tolerated by a normal individual, there are subjective complaints of fatigue, palpitation, dyspnea, pain in the chest, dizziness. The patient is seen to be apprehensive, trembling, breathing rapidly. The pulse is rapid, the palms of the hands wet with sweat. No evidence of organic disease of the heart or of any other organ can be made out. Thus by a process of exclusion the trouble is relegated to the class of functional affections, a neurosis, a classification that, as Wenckebach justly remarks, serves as a waste-basket into which we dump those diseases or disordered states concerning which our knowledge is still vague and obscure. We do not clarify our knowledge much by trying to explain it on the basis of endocrine disturbances, an explanation that is largely fanciful and lacking in scientific proof of its accuracy. Nor has any convincing demonstration been made as to perverted metabolism or as to the exact alterations in the workings of the nervous system, especially the vagus and the sympathetics, though vagal weakness or sympathetic hyperexcitability are referred to by certain writers as probable underlying factors. No worthy pathologic anatomy of this condition has been described. One is forced, therefore, to consider it from its clinical manifestations alone—a syndrome or symptom-complex rather than a clinical entity or a true disease—and to recognize it on the basis of the preceding history, the present subjective symptoms and the objective findings.

The cases for which an underlying organic lesion can reasonably be assigned as a cause are by nearly unanimous consent excluded from this category. Such are the cases in which an active tuberculosis can be made out, a myocarditis or valvular disease, or exophthalmic goiter. The irritable heart in these instances is regarded merely as secondary to, or symptomatic of, the organic trouble.

Two groups are generally recognized by writers, one a so-called constitutional group, the other the infectious. Not a few cases, however, fail to fit into either one of these classes. By the constitutional type is meant the one in which from childhood, often clearly by inheritance, there have been neurotic manifestations. The child has been delicate, perhaps an only child—the neighbors often say a "spoiled child"—solitary, shy, sensitive to criticism, perhaps an unusually good child who has not contracted the wild habits of the sophisticated worldly-wise bad boy, a child little inclined to vigorous athletic sports, to rough-housing and mingling with his fellows, one who is nervous, easily fatigued by exercise or by school work, and who in consequence has been anxiously watched over or petted by parents, conscientiously coached by teachers, but who is always a little weak physically and nervously, a little behind, an onlooker rather than an active participant in a normal child's life.

Some psychic insult, some great emotional strain, a sudden cardiac death in the family, a love affair, or some unusually severe physical or mental effort precipitates the attack with the symptoms just enumerated. War is the great and overwhelming cause that makes manifest this latent tendency in large groups at the same time. The condition then becomes, as it were, pandemic.

In the second group, past infections, particularly rheumatism, seem to have laid the foundation for the later neurocardiac outbreak. Many place great stress on this feature; others feel that the proof of the connection between an infection of years before and the present attack is not conclusivly shown. All agree that an acute infection such as rheumatism, chorea, pneumonia, influenza or trench fever is apt to be followed in one of this type by the syndrome of cardiac instability. DaCosta in his original article emphasized the fact that the irritable heart was prone to follow fevers and diarrhea, the diseases especially prevalent in the Civil War. These cases following infection, though they might be regarded as symptomatic, are generally classed with this group. A field is opened here whenever pathologic material is available, for study of the hearts of such patients especially for myocardial and nerve changes. There may possibly be some definite lesion or lesions, such as those described by Aschoff and others in rheumatism, that will enable us to place in a group by themselves some of these cases of irritable heart following hard upon the acute infections.

In order that the aims and rationale of treatment may be more clearly understood a brief sketch of the chief clinical features is added. Detailed descriptions may be found in the more elaborate articles, references to a few of which are given in the references.

Symptoms.—The commonest symptoms are pain, dyspnea, dizziness, palpitation, precordial tenderness, fainting. The pain which Hume found in 76 per cent of his series is referred to the chest, generally the left side; the patient usually says "in the heart." It is variously described as sticking, darting, aching. It rarely has the constricting character of a true angina though occasional radiation to the shoulder or arm may suggest such condition. The sensitiveness of the skin which is frequently found on examination may be remarked by the patient even before its discovery by the physician in the routine of examination.

Shortness of breath is described by nearly all. The patient often takes a deep sighing breath and calls attention to it as an illustration of the dyspnea. Under excitement the breathing may go to 50 or 60 per minute. With attention distracted from the heart to other things the breathing may drop to normal or may even be normal when some physical effort is made. In most instances it will be found that the supposed dyspnea is really tachypnea.

Dizziness is common. A patient may faint. Palpitation is usually very distressing. He complains of becoming tired, of lacking endurance. This has often necessitated the giving up of school, foregoing athletics, changing to a lighter occupation. Sleep is often poor, disturbed by dreams. He is unable to concentrate mentally. There is an admission of nervousness, introspection, fear of weak heart or sudden death. He calls attention to his trembling, to his sweating palms.

When such an individual is stripped for examination he is usually found to be flabby-muscled, his face pale or perhaps flushed, the vessels of the neck pulsating forcibly, the body shaking, the sweat trickling from the axillæ, and the palms of the hands wet and cold. He is evidently anxious and apprehensive, the voice trembles, he complains of pain in the left chest. The heart beats rapidly, perhaps 150 per minute during the examination, but may quickly drop to 90 or 100 as the ordeal is at an end. The apex impulse is diffuse with often a hint of a thrill. There is often a systolic whiff at the apex or a suggestion of a presystolic murmur, reminding one of mitral stenosis. There may be a splitting of the tone. Epigastric pulsation may be marked. The blood-pressure is apt to be a little above normal. The hopping test causes the heart's rate and the pressure to increase rapidly, but in all except the severer cases, after two or three minutes the pulse and the blood-pressure drop to the points noted before the test. One fact generally impresses the examiner, the small amount of physical variation from the normal compared with the numerous and marked symptoms.

Diagnosis.—The examiner before concluding that he is dealing with the effort syndrome pure and simple must exclude organic heart disease by the ordinary routine of physical examination. The systolic murmur should not be regarded as evidence of a mitral lesion unless the heart is enlarged, the pulmonic closure clearly accentuated and the exercise test poorly tolerated.

The X-ray may be of help in determining the size and shape of the heart. The electrocardiograph is of comparatively little help in this group of cases, though it may exceptionally detect some unsuspected flutter or other anomaly of conduction, more clearly establish the extrasystolic character of an irregularity or make plainer a respiratory sinus arhythmia or a paroxysmal tachycardia.

Tuberculosis, suggested by the fatigue, rapid heart action, flushed face, sweating and dyspnea, is by no means easy to eliminate. Careful examination of the chest, with the help of the X-ray, the search for bacilli in the sputum, temperature records, particularly after exercise, tuberculin tests, are all helpful. Suggestive facts in the past history should also be sought for, such as the history of previous pleurisy, joint inflammation or other possible tuberculous process.

The close resemblance of the irritable heart to hyperthyroidism has undoubtedly led to confusion of the two conditions. Some, in fact, regard thyroid overactivity as the underlying cause of both conditions, a belief not held by many who have studied large groups of patients with irritable heart. In this latter condition the thyroid is rarely much enlarged, exophthalmos is seldom seen, the tremor is coarse and is more of a shaking than a tremor, and the heart is of normal size. The pulse in sleep and in the recumbent posture becomes slower than in exophthalmic goiter, that is, it is not so persistently rapid no matter what the position of the body. Dyspnea and pain in the cardiac region are much more striking features. The metabolic rate is usually about normal. The patients with these two disease conditions are psychologically different. Peabody has well called attention to the fact that the hyperthyroid case is characterized by nervous activity, restlessness, physical and mental energy; he has to be held back, he rebels against the rest cure. The patient with the irritable heart is in a state of mental inertia, is desirous of evading even the strain of normal life; he has to be urged forward, he welcomes the suggestion of treatment by rest.

A fact not to be forgotten is that a patient may have a genuine organic disease of the heart such as a myocardial or a valvular lesion with neurotic symptoms added. This is frequently seen in patients with mitral stenosis where the rapid action of the heart, palpitation, sticking pains, call the attention of the sufferer to the heart, and where the directions of the physician as to the necessity of not overdoing add the element of fear that makes the patient decidedly neurotic. These cases must, of course, be considered in studying this group.

Prognosis.—The prognosis as to life is good. Permanent relief from symptoms, however, with the building up of physical and nervous strength to enable the sufferer to become an active and efficient member of the social body is a much more uncertain question. Especially in the so-called constitutional cases is the outlook discouraging. Many of these it was learned could never become efficient soldiers by any process of training. Some were found fit for light military service. The same differences are met with in civil life. Much depends on the inherited and acquired make-up of the patient, on his environment and on the way the doctor handles the case.

It is of this constitutional group that Robey and Boas were evidently thinking when they wrote: "We have become convinced that neurocirculatory asthenia is due to a fundamental nervous instability that cannot be cured." And again: "Real cure is impossible." Galli's expression "hereditary myocardism" has a ring of pessimism in it. Kerley, speaking of these constitutionally substandard children, says that thus they entered the world, thus they continue throughout perhaps a long life regardless of attempts at improvement.

Treatment.—One never knows how much is done by attempts at prophylactic treatment; one cannot tell when a child, for instance, is "threatened" with an irritable heart and whether the measures employed were the real reason why the condition did not appear. But in children or young adults who are of neurotic parents and in whom signs of "nervousness" begin to appear, something may be accomplished—at least the attempt should be made—by warning against too much coddling, by encouraging out-of-door sports and the mingling with other children, by training them so they may endure more of physical effort. So far as possible worries, psychic upsets, brainstorms should be avoided. There should be no hint of heart disease by doctor or parents; this is a not infrequent cause of a cardiac-nervous outbreak, from which time the individual's thought and acts will be centered about the heart. Convalescence from acute infections should be prolonged until the patient has fully recovered bodily and nervous strength. The pediatricians report that occasionally cases of irritable heart are seen in children where undernourishment or improper bringing up by the parents seems to be responsible causative factors. Both these conditions are avoidable.

The success of the active treatment will depend largely on the tact and firmness of the physician. If he receives the confidence of the patient, if he is a good judge of human nature, in other words, if he is a born practical psychologist, and if he is long-suffering and persistent he may work marvelous changes. This fact is recognized by practically all who have had experience in this type of disease. Wenckebach recognized it early in the War. Thomas states the facts clearly when he says: "A little psychotherapy does more than medicines." And again when he says it is necessary to persuade the patient of the innocuousness of his ills, to give him the desire to fight, to utilize to these ends all the intellectual and sentimental dialectic possible.

At times a tolerant listening to the tale of the sufferer, a thorough and painstaking examination with a positive statement that there is nothing organically wrong with the heart and an explanation of what really *is* the matter, together with an outline of treatment and a hopeful outlook for the future, will go a long way toward bringing about a cure. Some of these patients are timid and of a retiring disposition; they may be of the sissy or goody-goody type. It may sometimes be advisable to encourage these patients to loosen up a little, to be a little wicked, to ride in the smoking car perhaps, to utter a swear-word, or dance with a girl. It may prove to them that disaster does not necessarily follow what they thought were forbidden acts. Early masturbation, and the secret reading of sexual literature sent by advertising quacks may have caused unnecessary fears and great mental suffering to one who desires marriage but fears he is unfitted for that state. Reassurance on this score, perhaps leading to a happy marriage, may help some of these unfortunates.

Home influences can be corrected, at least the attempt can be made to correct them. The coddling and petting of the overwatchful mother can perhaps be stopped. A young man who came before me on an appeal to the District Board in the first draft of the War was a typical cardiac neurotic. With sweat running from every pore of his trembling body, and tears running down his cheeks he implored me not to send him into the army because he knew he could not sleep in a room or tent with other men, for from the day of his birth, twenty-three years ago, he had always slept in the same bed with his mother. Such conditions, unless corrected, will defeat every effort at cure.

Golf, tennis, baseball, dancing, billiards, bowling, swimming, walking should be encouraged. These sports should be entered into gradually, the one chosen being the one especially adapted to the endurance of the particular individual. The out-of-door sports are preferable. The benefit comes not alone from the bodily exercise and the fresh air but from rubbing elbows with others of the same age and of the same or the opposite sex. Home gymnastics as by dumb-bells, Indian clubs, setting up exercises, etc., while of value, are seldom persisted in, become irksome and are given up; the element of fun and companionship is lacking. Gymnastics of this type have been aptly called a form of solitary vice.

Much attention in the armies was devoted to systematic and graded exercises or drill. Sir Thomas Lewis obtained good results in this way, setting a standard for the British Army which was followed by many others. He used these drill exercises for a double purpose: as a test of the degree of a man's incapacity, that is, as a means of diagnosis and of classification. In this way it became clear that some men were utterly unfit for any kind of soldier's duty; others were suited only to light military work; others by training were brought up to the grade of full efficiency. For these latter, easy exercises were followed by more strenuous and then by long hikes. Others beside Lewis reported good results from this or similar plans. The Lian exercises in France were founded on the same principle. Some, however, as Robey and Boas were not as optimistic as to the end results. They felt that a good product could not be made from poor material. It is not possible for a 25 horse-power automobile to haul the heavy load that a 50 horse power truck can.

In civil practice it is difficult to control and force the carrying out of such exercises as was done in the army. Occasionally this can be done in a sanitarium. In selected cases this form of treatment should be tried where possible. Systematic graded exercise will do much to improve the appetite, relieve insomnia, quiet nerves, strengthen the muscles of the body including the heart muscle. It is gratifying at times to see how under this régime the tachycardia, palpitation, precordial pain, trembling and sweating disappear as a man regains muscle tone and self-confidence. The suggestive effect of this plan of treatment is one of its valuable features.

Meredith has reported on her success with girls in college and in factories by insistence on fresh air, good food and exercise. Not all our cases are cases that would accurately fit into this group of cardiac neurotics but some clearly belong there.

Baths, especially the cool morning shower or plunge bath, are often helpful.

Diet suited to the relief of constipation is far better than laxative drugs. Emphasis should be laid on cooked and raw fruits, coarse cereals, vegetables of all kinds, butter and cream.

Bad teeth with apical abscesses as revealed by the X-ray should be removed or treated by a competent dentist. Diseased tonsils may need removal. Infected sinuses, inflamed gall-bladders and all other tissues that are clearly foci of infection that may be causing or aggravating the condition should be given proper treatment. Radical and indiscriminate surgery, however, as is too often practiced on teeth and tonsils is to be deprecated. I have seen it definitely increase symptoms—it adds an element of traumatism to the existing neurosis.

Only rarely is a stay in a sanitarium advisable. Too often it merely intensifies the notion of invalidism in the patient. If the right man, however, is in charge, one who can treat the patient's mind as well as body, good results may be forthcoming. Rest cures, except in rare cases and then only at the beginning of treatment, are of little avail; they are generally harmful rather than helpful.

Drugs are advocated by very few of the many writers on this subject. Bromids have been occasionally advised, or a laxative, and once in a while a hypnotic. Drugs really serve to keep up the notion in the mind of the patient that he is ill and that there is some disease needing treatment. Digitalis is not indicated for a heart of this character.

One of the important objects is to fit the patients not alone for some kind of work but for the right kind of work. As Kerley says: "Expert occupational diagnosticians are needed." "There seems to be a certain level of strain below which these people can live without their symptoms, and their one effort in life is to keep below this level" (Wearn and Sturgis). To try to force them above this level is often disastrous. There are men and women who are clearly hopelessly unfitted—they are in the so-called constitutional class—for any strenuous work. If it is attempted they fail. To try to push them through college is a mistake. Any occupation demanding active work or great responsibility is beyond their powers. They naturally drift into some easy subordinate job which they perform indifferently at the best or else they become derelicts. Others by encouragement and training can do work of a higher order. The physician can often help fit the man to his job and the job to the man. Often the patient is being held by ambitious or misguided parents at some task to which he is ill-suited or which is distasteful to him. A change from the busy office or store of the city to the quiet out-of-door life of the garden, orchard, chicken farm or ranch may work wonders. And not a few girls who fret under the restraints of home do much better physically if allowed to go to work, earn their own living and mix with the world. There is, however, a large group of women, clerks, stenographers and teachers, who from long hours, indoor work of a sedentary character, no chance for exercise, with perhaps home cares and a love affair besides, are often dangerously near this symptom-complex. Some acute disease, a doctor's careless word about a murmur, some sudden mental shock and we have a full-fledged case of irritable heart, neurocirculatory asthenia.

REFERENCES

DaCosta, J. C. On Irritable Heart, etc., Am. Journ. Med. Sc., lxi, 17, 1871.

Galli. Il Cardiopalmo nei Soldati, Policlin., xxiv (Practica), 378, March 18, 1917; xxvii, 65, 1920.

Kerley, C. G. The Effort Syndrome in Children, Arch. Pediat., xxxvii, 449, 1920.

Lewis, Thomas. The Soldier's Heart and the Effort Syndrome, New York, 1919.

Meredith, Florence L. Functional Heart Disturbance in Women, Boston Med. & Surg. Journ., clxxxi, 734, 1919.

Oppenheimer, Levine, Morrison, Rothschild, St. Lawrence, and Wilson. Report on Neurocirculatory Asthenia and Its Management, Mil. Surgeon, April, June, 1918.

Peabody, Wearn, and Tompkins. The Basal Metabolism in Cases of the Irritable Heart of Soldiers, Med. Clin. North America, ii, 507, 1918.

Robey and Boas. Neurocirculatory Asthenia, Journ. Am. Med. Ass., lxxi, 525, 1918.

Thomas, A. E. Les Affections du système circulatoire chez les soldats des armées belligérantes. Revue méd. de la Suisse Rom., xl, 422, July, Aug., Sept., 1920. This contains good German and French Bibliography, 1914-1919.

Warfield and Smith. Journ. Am. Med. Ass., lxxi, 1815, 1918.

Wearn and Sturgis. Studies on Epinephrin. I. Effects of the Injection of Epinephrin in Soldiers with Irritable Heart, Arch. Int. Med., xxiv, 247, 1919.

Wenckebach, K. F. Uber die Behandlung Herzkrankte Soldaten in Kurorten und Heilstätten, Wien. med. Wchnschr., lxvii, 1926, 1917.

CHAPTER XV

THE TREATMENT OF CONGENITAL CARDIAC DISEASE

MAUDE E. ABBOTT

INTRODUCTORY

The special treatment of cardiac disease of congenital origin has received but scant attention in the past. Even in extended treatises on heart disease in childhood, it has usually been relegated to a final paragraph, and it has not until now been considered of sufficient importance to warrant a place in a System of Therapeutics.

The cause of this hiatus has lain in the relative infrequency of the cases of clinical import, the obscurity of their etiology and that of the cyanosis that forms their most characteristic feature, the futility of any remedial measures directed at removing the circulatory disturbances which they create, and especially in the widespread conception that has prevailed of the extreme gravity of the prognosis in the majority of the cases. It is worthy of remark that this gloomy view has been based, as pointed out by several recent writers, upon experience gained in the past chiefly in hospital practice, of those more complicated and severe cases which are brought to the physician for the relief of symptoms of deficient aëration setting in in the first weeks or years of life, and which terminate fatally, in most instances, before the age of five years.

The departure which this article thus represents is significant, we venture to think, of the marked increase in the general understanding of and interest in this subject that has taken place in the last decade, and which has resulted in a more favorable view of the prognosis, at least in that larger group of so-called non-cyanotic cases whose expectation of life is good, as well as in the recognition of the wide field for preventive and symptomatic treatment that exists both among these and also in those graver and more complex forms of *morbus cœruleus* where we may hope to alleviate though we cannot cure.

Several factors have taken part in this advance. The most important one, from the popular standpoint, is undoubtedly the routine examination of the cardiac area of normal and healthy individuals in late childhood and early adult life, which is carried on so extensively to-day through inspection for schools and colleges, and for military service both now and during the recent World War, and also the prolonged study of signs and symptoms through the establishment of cardiac clinics and the provision made for the care and after-care of "potential" and convalescent cardiac cases, both through the social service departments of our large hospitals, and through such organizations as the New York Association for the Prevention and Relief of Heart Disease. The observations made under these conditions, with the help of the newer methods of precision for the graphic presentment of cardiac irregularities, and in the light of the teaching of Sir James MacKenzie and his followers to the effect that the condition of the muscle rather than the character of the murmur is the criterion by which the heart's capacity is to be gaged, have effectually demonstrated to the profession that certain irregularities of the circulation, manifested by well-defined atypical cardiac signs may and do persist throughout life without interfering in any way with the normal activities of the subject, while yet presenting a certain menace that might well suggest precaution against undue risk.

Secondly, this small but fruitful corner of cardiac pathology shares in the illumination that has been cast upon the entire subject of the causation of congenital cyanosis, by the numerous studies that are now being carried out upon problems of the circulatory rate and respiratory exchange, diffusion of gases in the lungs and oxygen consumption in the tissues, and cognate subjects, that have recently culminated in the brilliant results of researches published by Lundsgaard and Van Slyke, by means of which we are enabled, for the first time, to make a *quantitative* analysis of each of the various factors that combine to produce the clinical evidence of oxygen unsaturation of the blood.

Lastly, it may fairly be conceded that the morphological evidence adduced by the statistical analysis of a large number of cases reported in the literature, with full details of clinical history and autopsy findings, such as has been carried out by Vierordt, Abbott and others, has had its share in the elucidation of this field, by supplying a basis from which conclusions of practical import can be drawn. Of these, the most important from the point of view of prognosis, and therefore of treatment, is the differentiation of two distinct clinical classes, a larger so-called non-cyanotic group, comprising localized defects of the cardiac septa, patent ductus arteriosus, and minor anomalies, in whom distinctive physical signs exist, but cyanosis is *absent, slight, transient* or *late,* and in whom the expectation of life is good; and another, somewhat smaller group, the cyanotic cases, comprising the right-sided valvular stenoses and atresias, transposition of the arterial trunks and complete absence of the cardiac or arterial septa, in whom the symptom-complex of congenital cyanosis is an early and prominent feature, while distinctive physical signs may or may not exist, and which either end fatally in the first weeks or months of life as extreme cases of *morbus cæruleus,* or progress slowly downward through the years to the same inevitable termination according to the degree and complexity of the circulatory anomaly.

In the author's series of 631 cases so analyzed, there were 325 of the non-cyanotic as against 233 of the cyanotic group, the remaining defects in the series rendering the subject non-viable or being for other reasons not of clinical import.

Fortmann, writing independently of our findings, in 1918, distinguished the same groups and stressed the importance, for clinical purposes, of differentiating between them, and several writers in the German literature since then quote his authority in drawing this distinction, which is, however, not new, either with him or the writer, having been drawn by Bamberger in 1857. The occurrence of the noncyanotic cases was recognized clearly in France as far back as 1879, when Roger established the clinical picture of localized uncomplicated defect of the interventricular septum that has since borne his name, on the basis of a case in which a rough systolic murmur and thrill with maximum intensity over the third left interspace existed, unassociated with any trace of cyanosis or other evidence of cardiac strain. In 1891 Dupré presented the first case of uncomplicated septal defect diagnosed during life on the basis of the characteristic physical signs as *maladie de Roger* and confirmed by autopsy, and insisted on the permanent and invariability of the murmur and the complete tolerance of the lesion. The occurrence, in this case, of cyanosis during the last months of life in the presence of an intercurrent bronchopneumonia is stated in this report but is not mentioned in his summary as a diagnostic feature. In 1889 Bard and Curtillet in a communication on the clinical picture of defects of the interauricular septum pointed out as characteristic of these cases the absence of symptoms during life and their termination by a late cyanosis (*cyanose tardive*), setting in during the course of a terminal bronchopneumonia, or other intercurrent event causing a rise of pressure in the right ventricle and a reversal of flow so that venous blood passed into the arterial stream through the defect. Reiss in 1892 pointed out, on the basis of a series of cases in the literature, that a late or intermittent cyanosis was characteristic of many cases of *maladie de Roger,* and drew the interesting conclusion that the cyanosis or *maladie bleue* in these cases was not the result of a simple cardiac septal defect, for the blood under physiological circumstances would go from left to right; but that in aged persons or those suffering from pulmonary trouble, such as tuberculosis or pneumonia, the pressure would become stronger than normal in the right ventricle which would therefore undergo hypertrophy and will send a current from right to left through the defect. LeHoux in an interesting thesis entitled Maladie de Roger, on the basis of these and other observations definitely adds the feature of a late or intermittent cyanosis to the symptomatology of interventricular septal defect.

Similar conditions to those in cardiac septal defects exist in patent ductus arteriosus, and here also the presence of physical signs with cyanosis absent, transient (in dyspneic attacks), or late, forms an eminently characteristic picture. In the author's series of 64 cases of patent ductus arteriosus analyzed, cyanosis was slight or transient in 3, "moderate" in 4, terminal in 10, and absent in 37; in no case was it "marked." Of 43 cases of uncomplicated defects of the interauricular septum, cyanosis was "slight" or "transient" in 4, moderate in 2, marked in 1, "terminal" in 13, and absent in 23. Among 37 defects of the interventricular septum analyzed, in 2 this condition was complicated by dextroposition of the aorta (cases by Simmons and Eisenmenger), and in one (Carpenter) by bicuspid pulmonary valves, and in these 3 cases cyanosis was "marked"; in the remaining 34 cases, cyanosis was "slight or transient" in 6, moderate in 2, terminal in 5, and absent in 21. In 27 out of the 37 cases, a loud systolic murmur was heard over the precordium, and in 9 of these cases this murmur was accompanied by a thrill.

The above considerations clearly indicate that in order to obtain a rational basis for the preventive and symptomatic treatment of congenital cardiac cases, certain points in their pathogenesis, classification and differential diagnosis must be briefly reviewed.

PATHOGENESIS

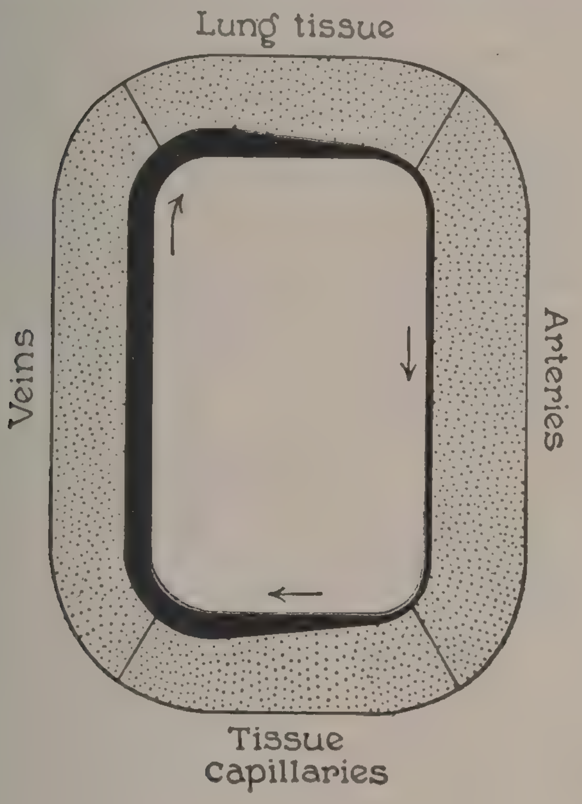
Congenital Cyanosis.—This term is applied to the characteristic symptom-complex of those cardiac defects which induce anomalous conditions of the circulation mechanically interfering with the due oxygenation of the blood. It consists in a bluish discoloration of the skin and mucous membranes, more marked in the peripheral parts of the body, varying in intensity with the degree of circulatory disturbance and associated in the more extreme and advanced cases with the other signs of deficient aëration, dyspnea polycythemia and cyanosis retinæ. These phenomena are so striking that they have excited observations from the earliest times; and ever since de Senac in his "Traité du Coeur" in 1749, in reporting the first fully studied case with autopsy, ascribed them to admixture of venous with arterial blood through a deficient interventricular septum, and Morgagni in 1761, in reporting a case of pulmonary stenosis, established as a rival theory the existence of venous stasis at the periphery, their pathogenesis has been the subject of debate and much confusion of thought. It is interesting to learn in the light of recent advances, that both these great observers were right in their recognition of the essential etiological factor at work.

We owe the remarkable elucidation which this obscure subject has just received to the genius of N. Zuntz, Haldane, Krogh, Barcroft, Van Slyke and their fellow-workers in the same field, very numerous especially of late years, in devising various forms of apparatus for the direct estimation of the oxygen and hemoglobin content of the blood, and to the researches of Lundsgaard and his associates, working at first on oxygen consumption by the nitrous oxid method of Krogh and Lindhard on the alveolar air, and, during the past six years, by the Van Slyke technic on the oxygen content of the venous blood in normal and disordered conditions of the circulation both in cyanotic and non-cyanotic states. As a result of the latter investigation, not only has the primary cause of congenital cyanosis been experimentally determined to be an increase of the reduced hemoglobin content to a point of known threshold value, but also, by a brilliant series of inductions, the relative magnitude of the influence of the various etiological factors has been quantitatively determined. For the full exposition of this fascinating subject, the reader is referred to the important monograph by Lundsgaard and Van Slyke to which reference has already been made.

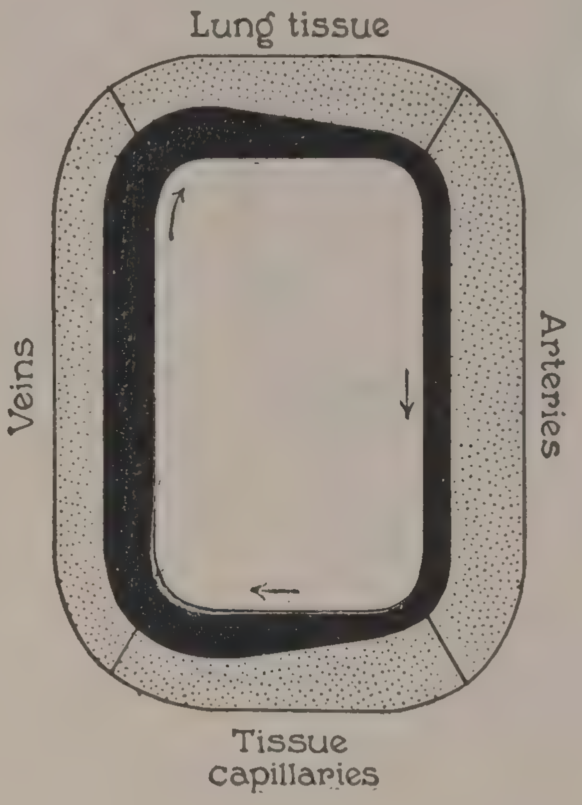
As a clear understanding of these etiological factors is essential to the appraisal of the possibilities, as well as the limitations, of the symptomatic treatment that is alone indicated in those tragic cases in which congenital cyanosis is a pronounced feature, the following brief outline, abstracted from the monograph in question is presented, together with several of the illustrative charts and diagrams with which the article is enriched, and which are reproduced by the kind permission of the authors.

Primary Cause.—The primary cause of congenital cyanosis, that is, the change in the blood itself that is responsible for the discoloration, has been proved in these investigations by a series of experiments carried on in cyanotic and non-cyanotic individuals, to be an increase of the reduced hemoglobin up to or above the "threshold value" of 6.5 volumes per cent of the total hemoglobin in the capillaries. In the same series of experiments, determination of the carbon dioxid and oxygen content showed that variations in these bodies had no causal relation whatever to this problem. For convenience of expression and notation, and for the reason that the oxygen values are those determined, the amount of "increased reduced hemoglobin" is expressed in terms of "increased oxygen unsaturation" which figures are obtained by subtracting the *oxygen content* (experimentally determined) from the oxygen capacity; and, since cyanosis is a descriptive term applied to the color of the skin as seen through the *capillary* walls, the degree of oxygen unsaturation is computed, and quantitatively expressed, in terms of "mean capillary unsaturation."

The following *normal values* have been established: (1) As the blood has an oxygen capacity of 20 volumes per cent of its hemoglobin, and as the blood issues from the lungs 95 per cent saturated, it follows that the *arterial blood* contains 19 volumes per cent of oxyhemoglobin, and under physiological conditions 1 volume per cent of reduced hemoglobin or oxygen unsaturation; (2) the oxygen-consumption in the tissues normally amounts by experimental determination to some 5 volumes per cent; this gives an *oxygen unsaturation in the veins of 6 volumes per cent;* (3) the "mean capillary unsaturation" is calculated by taking the average of the sum of the arterial and venous unsaturation (dividing it by 2, thus $\frac{1+6}{2}$); the result giving the normal "mean capillary unsaturation" as 3.5 volumes per cent. These features of the normal circulation are illustrated diagrammatically in the double rhomboid in Fig. 1, in which the amount of oxygen unsaturation physiologically present in the arterial blood, capillaries and veins, is shown by a black line of graduated width, increasing as these three different regions are traversed, and decreasing again as it passes through the pulmonary capillaries.



1



2

Fig. 1.—Diagram Showing the Proportion of Oxygenated Hemoglobin to Reduced Hemoglobin in Different Parts of the Circulatory System in Normal Resting Individuals.

Fig. 2.—Diagram Showing the Proportion of Oxygenated to Reduced Hemoglobin in Different Parts of the Circulatory System in a Case of Incomplete Oxygenation in Aërated Parts of the Lungs.

Black: fraction of total hemoglobin in reduced form. Dotted: fraction of total hemoglobin in oxygenated form.

(After Lundsgaard and Van Slyke "Cyanosis," from *Medicine,* 1923, with permission of Williams and Wilkins, Publishers.)

The "threshold value" of "mean capillary unsaturation" at which cyanosis usually appears is computed in the same manner from determinations of the arterial and venous oxygen content to be 6.5 volumes per cent.

Secondary Causes.—The threshold value of "mean capillary unsaturation" at which cyanosis usually appears is computed in the same manner as the normal values from determination of the oxygen-contents of the arterial and venous blood, and is 6.5 volumes per cent. A number of different factors may act either singly or in pairs or in more complex combinations to produce such an increase of oxygen unsaturation, or to modify the intensity or shade of color resulting from it. These factors are grouped as (*a*) *modifying factors,* which include alterations in color due to thickness of skin, pigmentation of skin or plasma, increase in number, width and surface of capillaries (important) or nature of the oxygen dissociation curve; and (*b*) *factors directly influencing the production of cyanosis,* by causing an increase of the oxygen unsaturation of the arterial blood.

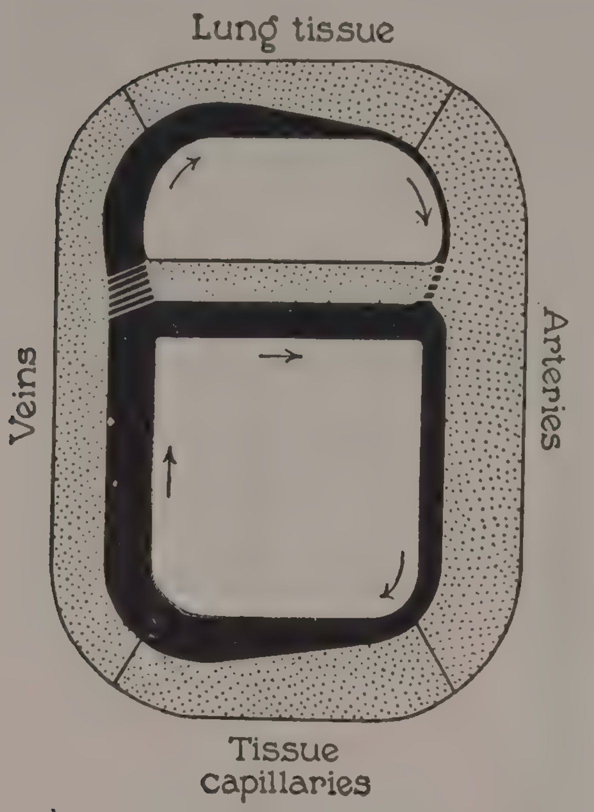


Fig. 3.—Diagram Showing the Proportion of Oxyhemoglobin to Reduced Hemoglobin in Different Parts of the Circulatory System in a Case Where a Fraction of Blood Passes Through Unaërated Channels from Venous to Arterial System. Black: fraction of total hemoglobin in reduced form. Dotted: fraction of total hemoglobin in oxygenated form. (After Lundsgaard and Van Slyke, "Cyanosis," from *Medicine,* 1923, with permission of Williams and Wilkins, Publishers.)

This latter group, *influencing factors,* being of direct etiological bearing, are of much greater importance and admit of numerical computation, a fact which adds to our understanding of their significance. Four such possible factors are distinguished as follows:

1. The oxygen unsaturation of the arterial blood may be increased beyond the amount which normally passes through the lungs unsaturated. This amount, estimated at one volume per cent, is called *"l"*; such an increase is brought about by a *decreased pulmonary oxygenation,* as in the lowered oxygen tensions seen in high altitudes, or the lessened oxygen diffusion through diseased alveolar walls, as in influenzal pneumonia (see Fig. 2). This factor has probably little or no bearing on cyanosis of congenital origin.

2. *Increased oxygen consumption in the tissues,* called "D." The loss of oxygen, which is normally 5 volumes per cent (3.5) may be increased to the threshold value by (*a*) raised metabolism, or (*b*) slowing of the stream in its passage through the tissues, as in the case of pulmonary

or mitral stenosis. This factor is the important one in the production of congenital cyanosis in right-sided valvular lesions.

3. *The total hemoglobin content of the blood* (symbol "T"). This factor cannot of itself produce cyanosis, but its increase, as in polycythemia, has a marked influence in raising the oxygen unsaturation to the threshold value at which cyanosis, from whatever cause, appears; and vice versa, in extreme degrees of anemia a very low hemoglobin content will prevent the appearance of cyanosis.

4. The fourth factor is in itself pathological. It consists in the passing of venous into arterial blood in abnormal conditions of the circulation, such as through a cardiac septal defect, under conditions of raised pressure in the right ventricle, or through an unaërated part of the lungs, as in pulmonary emphysema, pneumothorax, or complete bronchial obstruction; to this phenomenon, which they term a "right to left shunt," Lundsgaard and Van Slyke give the title α. This factor is of all the most serious, although it is calculated that the threshold value will not be reached, other factors being normal, until about a third of the venous blood has passed into the arterial circulation, or the full half of one lung has been cut off from the blood supply. In these cases oxygen therapy is, on the face of things, of no value. This condition of "right to left shunt" is shown in Figure 3.

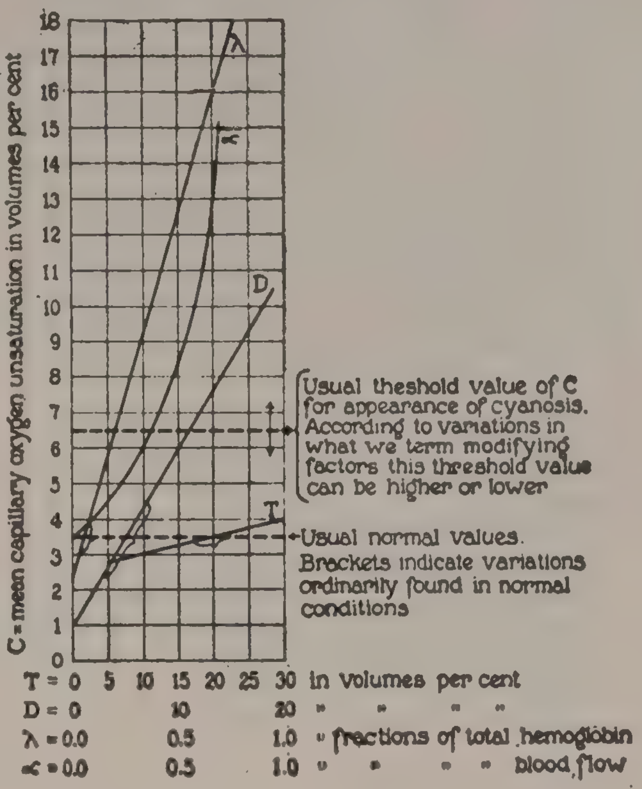


Fig. 4.—Diagram Showing the Relative Magnitude of the Influence on Mean Capillary Oxygen Unsaturation (C), of Variations in Total Oxygen Combining Power of the Blood (T), in Deoxidation Rate During the Passage of Blood Through the Capillaries (D), in the Fraction of Total Hemoglobin Passing Unoxidized Through Aërated Parts of the Lungs (λ), and in Fraction of Blood (α) Passing from Right to Left Through Channels to Which Air Has No Access. (After Lundsgaard and Van Slyke, "Cyanosis," from *Medicine,* 1923, with permission of Williams and Wilkins, Publishers.)

The value of these four factors has been experimentally determined, and their relative magnitudes to each other and to the normal and threshold values of oxygen unsaturation are shown in the form of curves in a very suggestive chart (Fig. 4).

Of even greater interest, especially from the standpoint of congenital cardiac disease, are the two remaining charts shown in Figures 5 and 6, where it is shown that these four factors tend to act in pairs, each to raise the threshold value of the other, *l* acting with *T* (decreased pulmonary oxygenation with increased hemoglobin content), and α with D (right to left shunt of venous blood with increased oxygen consumption from venous stasis). This latter combination, α and D, is the most startlingly suggestive, from the congenital cardiac standpoint, of the entire mass of information presented. It explains the marked cyanosis that invariably accompanies that commonest of all forms of congenital cyanosis reaching youth or early adult life, the cases of the so-called "tetralogy of Fallod," that is, pulmonary stenosis with interventricular septal defect and dextroposition of the aorta. Here, under the raised pressure in the right ventricle and in the abnormal position of the aorta, venous blood from the right heart communicates freely with the arterial stream (α shunt), and here also the conditions are fulfilled by a slowing of stream for a marked degree of increased oxygen consumption (+ D). On the other hand, the absence of an alpha shunt in those rare cases of pulmonary stenosis in which both auricular and ventricular septa are closed, and in which the only factor present is slowing of the stream (+ D), supplies the explanation why the cyanosis is here relatively *late* and *slight.*

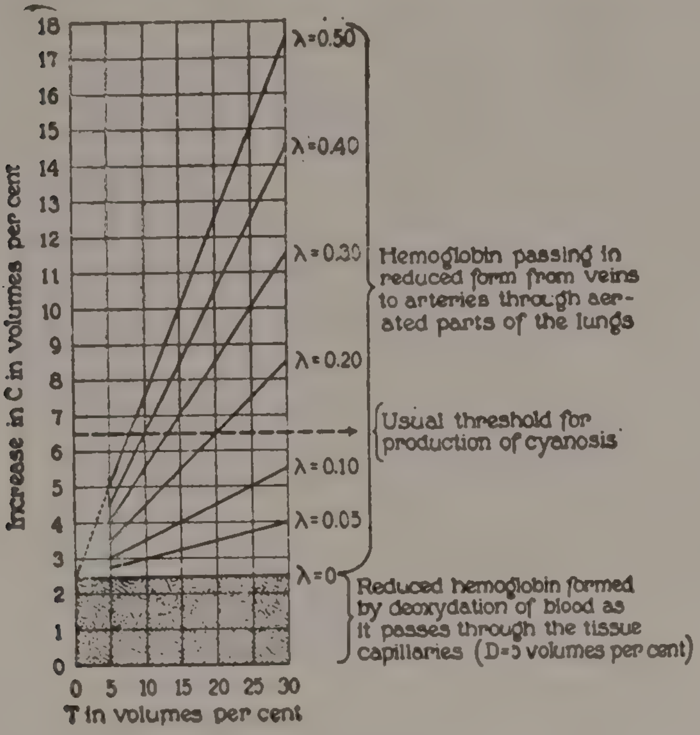


FIG. 5.—DIAGRAM SHOWING INFLUENCE ON THE MEAN CAPILLARY OXYGEN UNSATURATION (C) OF SIMULTANEOUS VARIATIONS IN T AND λ. (After Lundsgaard and Van Slyke, "Cyanosis," from *Medicine,* 1923, with permission of Williams and Wilkins, Publishers.)

Thus these two factors, revealed to us three centuries ago as the "admixture of currents" of de Senac, and the "venous stasis" of Morgagni, have to-day come into their own in their recognition, in terms of Lundsgaard's and Van Slyke's alpha and D factors, as the two "influencing" secondary causes of the increased oxygen unsaturation of congenital cyanosis, which may act singly, but more often, and practically always in severe cases, are conjoined. To them, in all such marked and advanced cases, are associated as "modifying" or subsidiary factors, an increase of total hemoglobin, supplied in the constant polycythemia, and the widened surface area of the surface capillaries, indicated by clubbing of the extremities, that complete the symptomatology, and fall into a rightly subordinate place in the etiology of the disease.

The Cyanose Tardive of the So-Called Non-cyanotic Group.—The above discussion of congenital cyanosis refers to conditions in the so-called cyanotic group of cases, especially to those marked cases in which Lundsgaard's and Van Slyke's alpha and *D* factors combine in those severe cases which have sooner or later a fatal termination. Of far greater interest from the standpoint of treatment are the conditions in that much larger group in whom cyanosis is absent or late and whose pathologic physiology and cardiac efficiency it behooves us to understand if we are to supply them with an adequate preventive and hygienic regimen that may preserve to them their full "expectation of life."

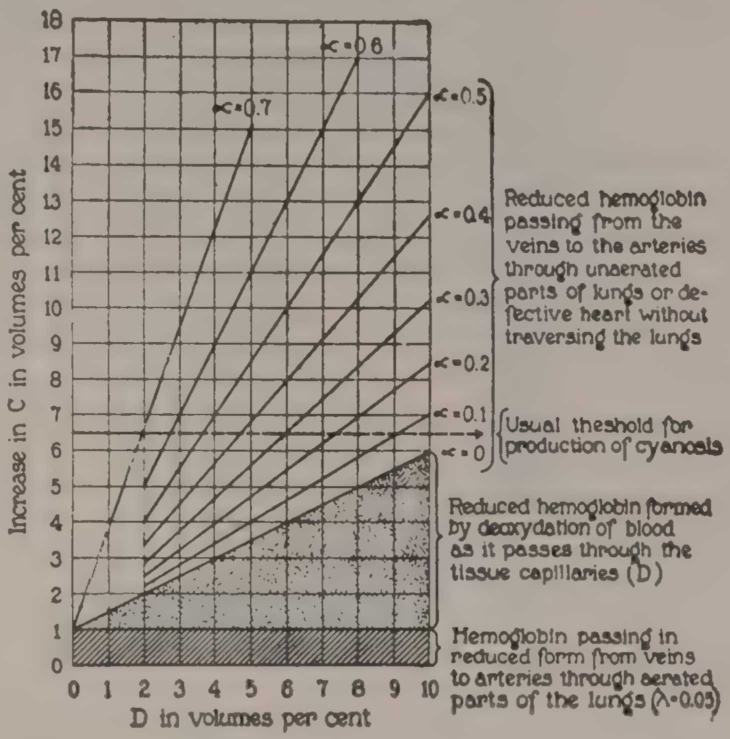


FIG. 6.—DIAGRAM SHOWING THE INFLUENCE ON THE MEAN CAPILLARY OXYGEN UNSATURATION (C) OF SIMULTANEOUS VARIATIONS IN D AND α. (After Lundsgaard and Van Slyke, "Cyanosis," from *Medicine*, 1923, with permission of Williams and Wilkins, Publishers.)

Such cases, as pointed out above in the introductory statement, are those in which a *localized* defect of the cardiac or arterial septa, or a patent ductus arteriosus exists, uncomplicated by conditions which would cause a rise of pressure in the right ventricle; for here, as has been pointed out by all students of this subject during the last fifty years or more, there exists under the relatively raised pressure that normally exists in the left ventricle a *left to right shunt through* the defect, arterial blood passing through it into venous, and causing indeed some undue loading of the pulmonary circulation, but supplying no cause for cyanosis, so long as these functionally normal conditions exist. When, however, the circulatory conditions are reversed and a rise of pressure in the right heart occurs, either through pulmonary obstruction or weakness of the left ventricle, as in aged persons, the pathological conditions of the right to left shunt are instituted, and we have the phenomenon of a "late" cyanosis, setting in as soon as the admixture of venous with arterial blood brings oxygen unsaturation to its threshold value. In such cases a slowing of the blood-stream from lowered cardiac efficiency is frequently an associated condition, and this again adds the effect of an increased oxygen consumption to the primary factor. To this condition of left to right shunt, which may be considered as physiological under the circumstances, we may give the symbol β (*beta*). A means of its quantitative determination is not yet established, for all the attempts of Plesch, Boothby and others to establish a satisfactory diagnostic method for the estimating of the amount of blood in the pulmonary as compared with the systemic circulation, have so far proved unsatisfactory. Lundsgaard's and Van

Slyke's diagrammatic representation of this left to right shunt is shown in Figure 4, which is to be compared with their diagram of the right to left shunt, reproduced as Figure 3.

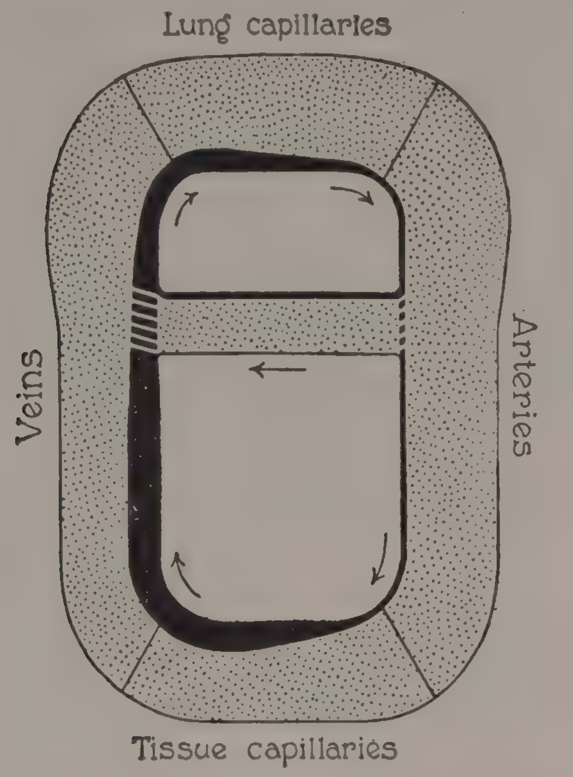


FIG. 7.—DIAGRAM SHOWING THE PROPORTION OF OXYHEMOGLOBIN TO REDUCED HEMOGLOBIN IN DIFFERENT PARTS OF THE CIRCULATORY SYSTEM IN A CASE WHERE A FRACTION OF BLOOD PASSES FROM THE LEFT SIDE OF THE HEART TO THE RIGHT SIDE THROUGH AN ABNORMAL COMMUNICATION. Black: fractional total hemoglobin in reduced form. Dotted: fractional total hemoglobin in oxygenated form. (After Lundsgaard and Van Slyke, "Cyanosis," from *Medicine*, 1923, with permission of Williams and Wilkins, Publishers.)

Figure 8 presents four other diagrams of the conditions shown by Lundsgaard and Van Slyke (Figs. 3 and 7 of this article), treated from another angle, as it were, namely, the content of the pulmonary blood supply in these various conditions. In I of these diagrams the heart and great vessels in the normal circulation are shown; in II the same with a left to right shunt, through a "compensated septal defect" (*cf.* Fig. 7); in III a right to left shunt (α) through a decompensated septal defect in which there is a raised pressure in the right ventricle without other anomaly; in IV also a right to left shunt of greater volume than in III through a ventricular septal defect in the presence of pulmonary stenosis, which has produced a greatly increased rise of pressure in the right ventricle (*cf.* Fig. 3).

CLINICAL CLASSIFICATION OF CARDIAC DEFECTS

A grouping of cardiac defects from the standpoint of their clinical significance and symptomatology is essential for practical purposes and as a basis of rational therapy. We may recognize the following:

1. A so-called *cyanotic group*, in which the symptom-complex of congenital cyanosis is an early and pronounced feature. These may be again subdivided into:

a. Those in which *cyanosis is extremely marked* and the conditions for an increase of oxygen unsaturation so grave that an *early fatal termination* is inevitable. Such are in the first place complete transposition of

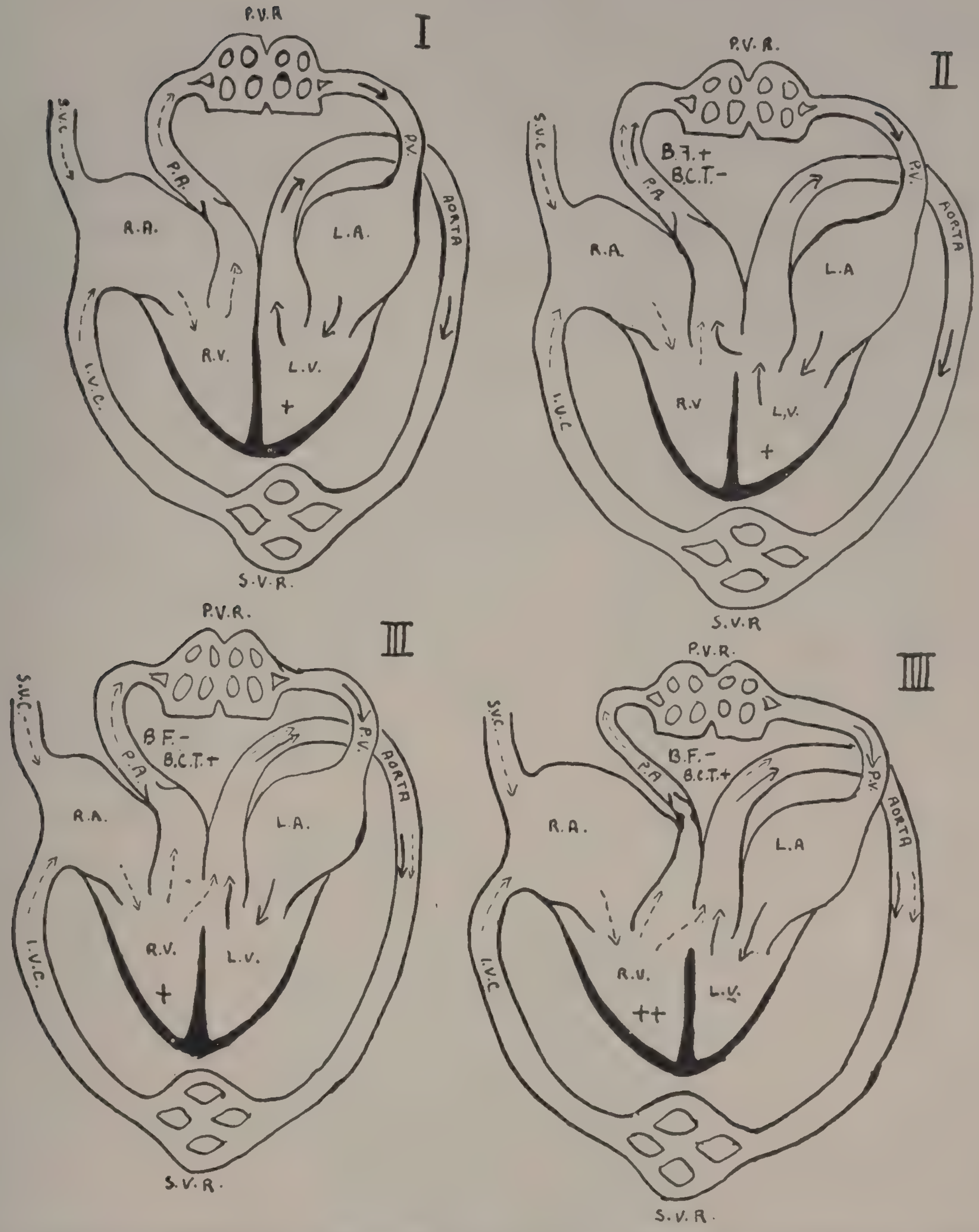


Fig. 8.—Diagrams Showing Inverse Ratio in Basal Carrying Tension and Volume of Blood in Pulmonary Circulation in Septal Defects. (W. M. Boothby and M. E. Abbot. Diagram by Louis Gross.)

P. V. R. = pulmonary venous reservoir; *S. V. R.* = systemic venous reservoir; *B. F.* = blood flow; *B. C. T.* = basal carrying tension.

I. Normal conditions. (*Cf.*, Fig. 1, showing Lundsgaard and Van Slyke's diagram of the same condition.)

II. Ventricular septal defect under physiological conditions of functional efficiency. The pressure is + in the left ventricle and a current passes from left to right through the defect, the blood in the pulmonary circulation artery is increased, the basal carrying tension (or gas load) is therefore (by a physical law) reduced. (*Cf.*, Fig. 7, Lundsgaard and Van Slyke's diagram of left to right shunt.)

III. Ventricular septal defect in cardiac decompensation or rise of pressure in the pulmonary circulation from any cause. The pressure is + in the right ventricle, the current passes from right to left through the defect, the blood in the pulmonary circulation is reduced. (*Cf.*, Fig. 3, Lundsgaard and Van Slyke's diagram of right to left shunt.)

IV. Ventricular septal defect complicated by pulmonary stenosis. The pressure is *greatly* increased in the right ventricle. (*Cf.*, Fig. 3, an exaggerated case of right to left shunt.)

the arterial trunks, pulmonary, mitral and tricuspid atresia, biloculate heart; persistent truncus arteriosus (common arterial trunk).

b. Those in which the symptom-complex of *congenital cyanosis,* though still present in its complete form, is less extreme, the cases *lasting to early adult life,* as in pulmonary stenosis with defect of the interventricular septum and dextroposition of the aorta (Fallod's tetralogy).

c. Cases of *congenital cyanosis of a still more moderate* though yet pronounced type, which may last on *into late middle life,* and frequently die from intercurrent complications. These are triloculate heart and those rare cases of valvular stenosis of the right heart unassociated with cardiac septal defect.

2. The so-called *non-cyanotic group,* in which cyanosis is absent, slight, or late. These also may be subdivided into:

a. Cases of *cyanose tardive,* in whom a septal defect is present and the conditions exist for the development of a late or transient cyanosis, provided circumstances intervene which will induce a rise of pressure in the right ventricle or in the pulmonary circulation, such as bronchopneumonia, etc. In these cases there exists a localized defect of the interauricular, interventricular, or aortic septum, or a patent ductus arteriosus without other complication, through which a left to right shunt occurs until some event supervenes which reverses the flow and projects venous into arterial blood. In all these conditions, characteristic physical signs are usually present upon which a diagnosis can be based, but symptoms are ordinarily absent until the onset of dyspneic attacks or a terminal cyanosis;

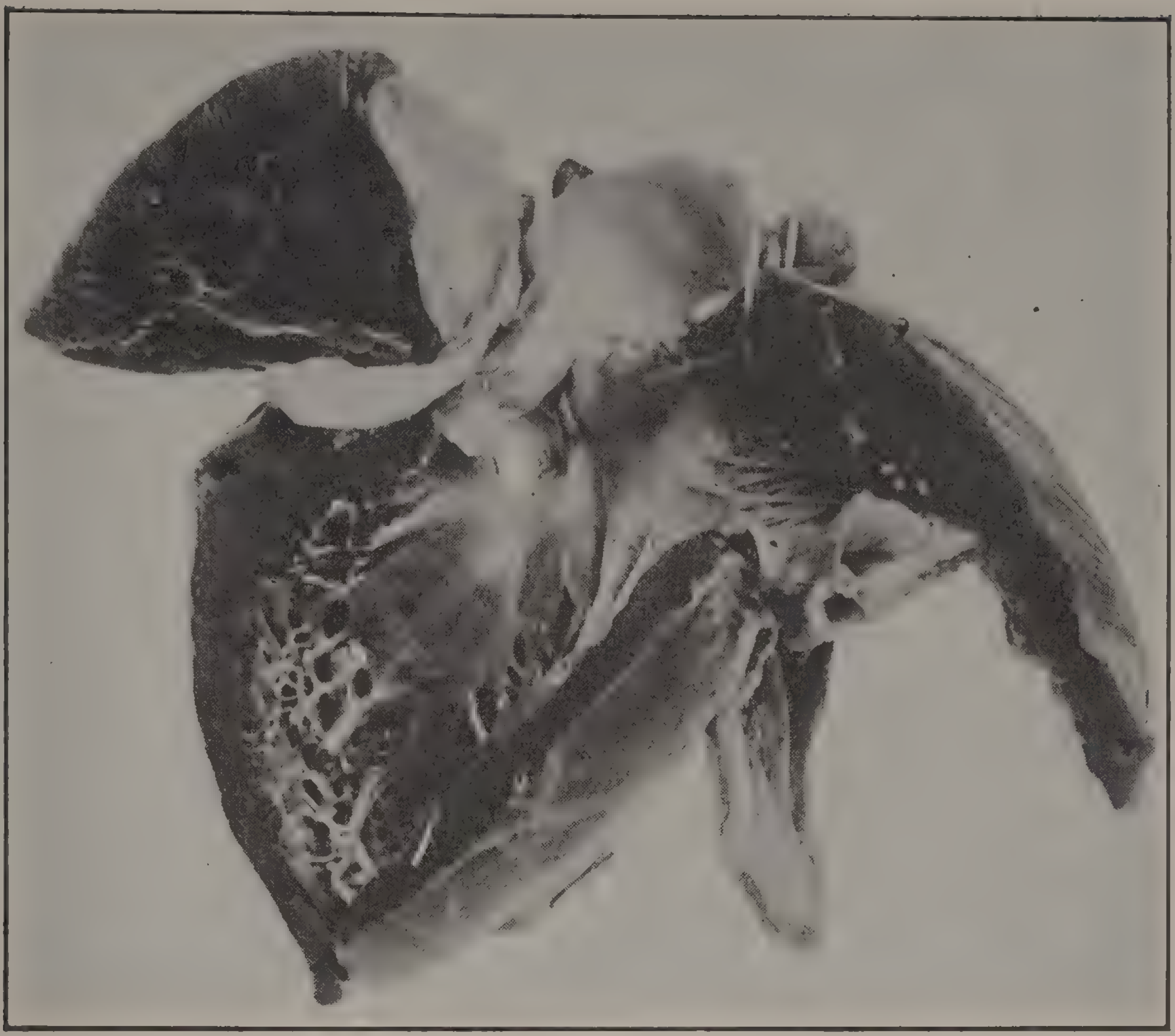
b. This comprises a motley group of *cardiac defects which* cannot of their nature interfere with oxygen unsaturation, but which may nevertheless be of actual clinical significance; such are pericardial defect, dextrocardia due to arrest of development, bicuspid or supernumerary semilunar valves, left-sided fetal valvular lesions, coarctation and hypoplasia of the aorta, and anomalies of the great arterial trunks at their origin from the arch. All these may in one way or another interfere with cardiac efficiency, or supply a seat of lowered resistance for an intercurrent infection.

3. The third group consists of those conditions which are:

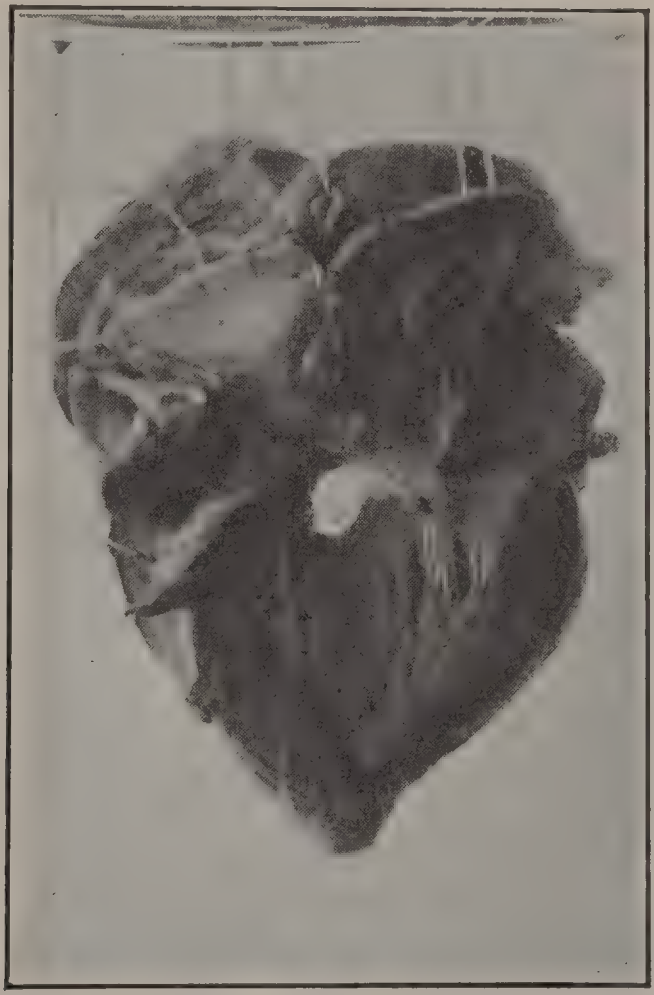
a. *Not in themselves of any clinical significance,* such as mirror picture dextrocardia, etc.

b. *Of so serious a nature that the infants are non-viable,* for example, ectopia cordia.

This is not the place to enter into a discussion of the differential diagnosis of these various defects. It suffices to point out here that, for intelligent treatment, the practitioner should possess a sufficient knowledge of this subject to permit of his recognition of the congenital nature of the



A



B

FIG. 9.—LOCALIZED DEFECT OF THE INTERVENTRICULAR SEPTUM IN TWO CASES BELONGING THROUGH THEIR ANATOMICAL CONFIGURATION RESPECTIVELY TO THE (a) CYANOTIC, AND (b) NON-CYANOTIC GROUP. From two specimens in the Pathological Museum of McGill University. Presented respectively by Dr. G. Lomer and Dr. John McCrae. A. Ventricular septal defect with dextroposition of the aorta which arises from both ventricles above the defect, receiving both venous blood from right and arterial blood from the left chambers (right to left shunt). Child, aged 4, cyanosis and clubbing. B. Localized septal defect. Aorta in normal position, entirely from left ventricle. Case of left to right "beta" shunt. No cyanosis, systolic murmur and thrill over middle of precordium.

lesion, and of the clinical significance of the so-called cyanotic and non-cyanotic groups.

TREATMENT

The discussion of this subject falls under the following heads:

Prenatal care includes the care of the mother during her pregnancy and before it, especially as regards laws of sex hygiene, and the care and prophylaxis in both parents of infective processes.

The treatment of the cyanotic group, by the alleviation of the distressing symptoms of oxygen-unsaturation, and the retardation, in so far as this is possible, of the downward trend of the disease.

The treatment of the non-cyanotic group involves the maintenance of the maximum functional efficiency within the limitations of the patient's cardiac reserve, by a carefully regulated personal and social hygiene, and the prevention both by these means and by the avoidance of pulmonary disease of a late cyanosis which transfers these cases into the cyanotic group.

Surgical measures both for the prevention of intercurrent complications by the removal of foci of infection and for the radical cure of the condition in the few cases where the mechanical conditions appear to render this possible or feasible.

Prenatal Care

For an intelligent discussion of this subject, our knowledge of the obscure etiology of cardiac defects must be briefly reviewed. Only in a relatively small group does the proximate cause lie in a fetal endocarditis running its course before birth and producing a valvular stenosis or atresia of the orifices, usually of the right heart. The great majority of cases, as Meckel pointed out in 1812, and as Rokitansky, Keith and others have fully demonstrated since then, are due to an arrest of development occurring before the cardiac septa are closed. The anomaly resulting from such an arrest differs, of course, according to the stage at which it occurred, the particular area it has involved, and the secondary changes that have resulted from it in other parts of the heart in the subsequent growth of the organ. For the understanding of the pathological anatomy of the particular defect, a knowledge of the facts of embryology is therefore essential, and in the light of the facts so revealed the most complicated anomaly becomes reduced to its simplest terms. A detailed account of the development of the heart with illustrative diagrams is given in the various monographs on the subject by Vierordt, Herxheimer, Abbott, Hirschfelder, and others, and in the great work of Rokitansky, and to these the interested reader is referred.

A subject of direct interest from the point of view of treatment is the remote cause of the defect underlying the arrest of development in any particular case. This question is still an obscure one, that cannot yet be said to be definitely solved. Statistics of associated anomalies in the same individual and in different members of the same generation, together with other facts, show, however, that cardiac anomalies are not, like polydactylism or hemophilia, ancestral traits which recur through succeeding generations. Hereditary predisposition takes but a minor part, and the immediate cause of the arrest lies, at least in the majority of the cases, in some process of infection, trauma, or faulty metabolism, acting within the maternal tissues or in the embryo itself or its envelopes, before the cardiac septa have closed in the eighth week of fetal life. This critical period is thus the only time at which prophylaxis of cardiac defects, in the sense of avoidance of the arrest of development which they portray, is possible. The indications lie not only in the judicious care and treatment of the mother during these weeks, but also in the exercise of a wider preventive hygiene in the case of both parents with a view to the successful conduct of future pregnancies, both by the vigorous treatment of infections, and by the observance of well-defined laws of eugenics and sex hygiene, the transgression of which may take an obscure but none the less active part in such results. An excellent illustration of some of the remoter elements involved in the etiology of the latter more obscure cases is supplied by the history of a specimen in the McGill Museum, presented by the late John McCrae, of pulmonary atresia with "incomplete heterotaxy" (situs inversus of the viscera and of the auricles of the heart with reversed torsion of the great trunks) from a case dying on the forty-ninth day with extreme *morbus cœruleus,* which was *the fifteenth child of a 46-year-old mother who was mentally deficient and had a harelip!* The inefficient metabolic processes of this elderly individual, herself of low mental and physical caliber, and laboring under the strain of the last of many pregnancies, evidently combined here to produce an "exhaustion-state" of the procreative powers that would appear to have been at least one important element in the causation of the defect. Similarly, there is considerable statistical evidence to show that marriages of close consanguinity, which apparently transgress certain obscure immunilogical principles, are liable to be followed by defects in the special senses, and possibly also in the heart, and are, therefore, to be condemned. So also, unions involving great physical disparities and undertaken in defiance of Nature's principles of selection are to be avoided.

Of importance also in the pathogenesis is the effect, during the critical eight weeks, of those obscure psychic influences that undoubtedly exercise a potent effect, whether through the action of internal secretions or some other organic path as yet little understood, on the processes of growth. Physical abuse of the mother by the drunken father of a child during this period of its embryonic life has been followed, in at least two recorded instances within our knowledge, by cardiac defects which have repeated themselves from a like cause in other children of the same generation. The direct trauma was here certainly the least part of the offense. And the converse, with its corollary, is also true. For, as Stockard has shown in his epoch-making contribution to the etiology of monstrosities, interference with the "rate of growth" at any given point results in its permanent retardation, and in that asymmetry of finer or coarser details, which, at one end of the scale creates ugliness or disharmony of feature, and, at the other, anomalies and finally monstrosities. In the establishment of those influences which most conduce to the harmonious and equable development that embryonic life demands for its normal fulfillment in a sane mind and a healthy and beautiful body, a fundamental obligation rests upon *both* the individuals who together share in the copartnership of responsibility that parenthood involves.

Pre-natal Infections and Intoxications.—In addition to the avoidance of such obscure and remote causes as are enumerated above, attention must be directed to the prevention or cure of direct noxa in the form of toxins or infective processes, which may exist in either parent, but which are especially serious in the mother, both before and after conception, and which may exert a directly inhibitory effect on the developing embryo. The most important of such noxa in the mother, at least as regards the congenital valvular lesions of later fetal incidence, would at first sight appear to be *rheumatic fever,* both because this is the commonest cause of heart disease in pregnancy and because the lesion of fetal endocarditis closely resembles the simple verrucose form of rheumatic origin in the adult heart, and differs, at least in the majority of the cases, both in character and distribution of vegetations from the sub-acute bacterial type so clearly described and demonstrated by Libman, and recently by Blumer, whose statistics show also that the latter more virulent infection is relatively uncommon in early childhood (and therefore probably in utero). Further investigations on this point are needed, and microscopic examination of the fetal myocardium in recent inflammation of its valvular endocardium should be made to determine the presence or otherwise of Aschoff bodies. The remarkable family incidence of rheumatic heart disease, and the preponderance of rheumatism as a cause of heart disease in pregnancy have been demonstrated with growing clearness by the work of Lawrence, Paul D. White and others, and direct transmission of the disease from the mother to the fetus in utero has been established by a number of observations, as in the finding by Poynton and Paine of vegetations on the mitral valve of an infant dying on the second day of life, whose mother had suffered from acute rheumatism in the later months of pregnancy.

Of other acute infections a history of tuberculosis, especially in the mother, occurs not infrequently in cardiac defects. Albasanz cites three such cases, and insists upon the importance of this factor, claiming that it acts by producing specific or sclerotic lesions, or by leading to a dystrophic state which favors the influence of even slightly toxic causes.

Small-pox in the pregnant mother was associated with pulmonary stenosis of the inflammatory type (ventricular septum closed) in a child born in the seventh month, which itself sustained an attack of this disease two weeks after birth, in a case reported by Niergarth.

Alcohol Intoxication.—The recent remarkable observations published by Dr. Stockard, on the effects upon the germ cells of animals exposed to the fumes of alcohol as seen in the later generations of large series of guinea pigs, demonstrate clearly the fact that such noxa in the parental organism is capable of exerting a directly inhibiting and deforming effect upon successive generations, so that, whatever conclusions one may draw upon the ultimate result of such intoxication in eliminating degeneracy in the race, the conclusion is inevitable, that malformations in the individual must frequently be attributable to alcoholic and other intoxicating habits, which are the direct cause of many cardiac defects.

Preventive Therapy.—The remarkable success that has attended the introduction of organized measures against rheumatism at Bellevue Hospital and elsewhere during the past five years, as shown by Lambert's statistics, make the indications for prenatal treatment in this connection very plain. The "expectant" or pregnant mother is to be shielded from the invasion of all such infections by every available means, both by the maintenance of her personal resistance and by the judicious treatment or removal of all possible foci of infection in teeth, tonsils, genitalia or elsewhere which might serve as portals of entrance for rheumatic or other poisons. Scrupulous oral and dental hygiene is especially imperative. Tuberculosis should be similarly guarded against, and pulmonary complications sedulously avoided. An active tuberculous lesion should debar the patient from motherhood at least until the quiescent stage is reached. Active tuberculosis in the father should be treated by his segregation until the infective condition is past.

Among prenatal infections *congenital syphilis* probably takes first place as an etiological factor in cardiac defects due to early arrest of development. This opinion is based on the frequency of a luetic parental history and of multiple associated anomalies in these cases, and on the researches of Warthin who has demonstrated a diseased myocardium in every case of hereditary lues he has examined. This view of the causal relation of congenital syphilis to many cases of grave cardiac defects cannot, however, as yet be said to be proved, for statistics based on autopsy findings of the incidence of cardiac anomalies in this disease are still lacking, and serological evidence is usually negative, so that many leading pediatricians unite in disclaiming any causal connection. A luetic history in one or both parents is certainly frequent. It was present in 23 out of

33 cases investigated by Steffano and in only 5 of his series could it be positively excluded. He insists upon the importance of vigorous prenatal treatment of the mother on this account and the treatment also of infants with cardiac defects where congenital syphilis is suspected. The common occurrence of multiple anomalies with or without a clear history of syphilis, or repeated miscarriages, is also evident from the literature and is frequently ascribed by the writers to the luetic history. In illustration might be quoted the case cited by l'Abbé of "pure" mitral stenosis associated with dwarfism and dental and palatal deformities in a patient with a history of paternal syphilis (general paralysis) and polylethalism of brothers and sisters in early life; and that reported by Queyrat, Gandry and Delinquand of congenital cardiac disease, in a case of vitiligo with facial asymmetry and malformation of the dental ramus and palatal arch, in which hereditary syphilis was claimed as the cause by the authors, although they were unable to demonstrate its presence by the usual tests. The frequent association of cardiac defects with Mongolian idiocy is also highly suggestive, Dr. John Thomson's statistics in this connection giving 28 cardiac defects in his series of 138 cases.[1] Here a syphilitic origin is doubtful on account of the frequent occurrence of mongolism in the last of many pregnancies or in very young primiparæ (indicating an "exhaustion-state" of the procreative faculties), and the curious phenomenon of its appearance in only one member of a heterologous twin pregnancy (suggesting a primary lesion of the germ-plasm), and laboratory evidence in these cases is also conflicting or misleading, as seen from the study by Stevens and its refutation by McLelland and Ruh. But again the peculiar Mongolian syndrome, with the typical facies and regularly associated deformities, together with the very early stage at which the cardiac defect has usually occurred (*persistent ostium primum*) strongly suggest an inhibitory action of disordered internal secretions or other little understood metabolic processes, the seat of which lies in the first place in the maternal tissue. Swanberg and Haynes conclude a full study of this subject of mongolism in twins with complete review of its bibliography, with the words, "the opinion of virtually all who have studied it is that it is due to *something that interferes with prenatal development, and that the adequate cause is to be sought in the condition of the mother during pregnancy."* It is hard to disprove congenital syphilis as a cause of cardiac defects. Warthin, whose large experience constitutes him an authority, believes that the ordinary tests for hereditary lues have no significance in childhood. His own histological researches on "Congenital Cardiac Syphilis" are of great interest. In 12 cases, all dying from unknown

---

[1] In a letter received by the writer after this article was in proof, Dr. Thomson writes: "I have notes of 300 cases of Mongolism at various ages. Those of them that were under five years of age when seen had symptoms of congenital cardiac disease in the proportion of 1 in every 6. In the older ones the proportion was 1 in every 8.

causes, the heart muscle was found to be the seat of an extensive diffuse or localized syphilitic myocarditis with spirochetes massed at the most advanced foci of degenerative and inflammatory change. Nine out of the 12 were infants or young children, but 3 were subjects past the age of puberty, aged respectively, fifteen, eighteen and twenty-two years. All were distinctly underdeveloped, and in one, a girl aged eighteen, marked infantilism existed. The oldest patient, a young woman student of twenty-two, whose father was in the last stages of locomotor ataxia, gave a history of having been a "blue baby" and of cardiac symptoms from infancy, and died from a bacterial endocarditis engrafted on a diseased myocardium. In a later communication by the same author upon the autopsy findings of two cases of congenital syphilis in infants with marked clinical manifestations, the heart muscle was found microscopically to be loaded with spirochetes massed together in large focal areas of advanced fatty and myxomatous degeneration and inflammatory cell proliferation, while all the other organs of the body were entirely free from spirochetes. The conclusion is drawn that congenital cardiac syphilis of the non-gummatous type is relatively frequent, and the interesting conception is formulated that in hereditary lues the heart is the usual seat for the colonization of spirochetes.

This entire subject of an etiological relationship between congenital syphilis and that deep-seated class of anomalies in which the more serious cardiac defects belong is thus still plunged in obscurity, and further investigations are needed, both into the relative frequency of anomalies of the heart in such cases of hereditary lues as come to autopsy, and, on the other hand, into the presence or absence of spirochetes in the heart muscle of grave cardiac anomalies.

The point to be insisted upon here is that, since an etiological relationship exists in certain cases, and in view of the remarkable success that attends antiluetic treatment of the expectant and pregnant mother, precautionary measures and rigid inspection and serological and other investigations should be undertaken, both of the expectant mother and of those infants which are the subjects of cardiac defects, with a view to vigorous antisyphilitic measures if these are indicated. To this end it is essential, first, to forbid marriage of all infected persons until at least two years after a reported cure (Holt); secondly, to establish in all suspected cases the existence or otherwise of the disease in the mother by a thorough physical examination and by the Wassermann test carried out first in the blood, and then, if this for any reason is doubtful, on the cerebrospinal fluid. In the event of a positive or even probable diagnosis of maternal syphilis, treatment must be at once begun both by intravenous injections of arsphenamin and by mercurial remedies. The routine carried out at any first-class clinic may be taken as a guide and is fully described elsewhere in this system. The kidneys must of course be watched for effects of arsphenamine poisoning, and mercury must not be pushed to the point of salivation. Such medication is not only well borne by the early embryo, but has an actively protective or curative effect upon it, statistics showing that in the great majority of cases the child of a syphilitic mother so treated in her pregnancy is born healthy, without luetic taint, whereas we know that practically all children born of mothers in the active stages of the disease, and who are as yet untreated, present unmistakable evidences of congenital lues. E. A. Morgan's statistics from the Toronto Hospital for Sick Children show 93 per cent of healthy offspring from syphilitic mothers who have been treated along similar lines.

TREATMENT OF THE CYANOTIC GROUP

Under this heading is considered the treatment, in so far as this may be feasible, of the characteristic symptom-complex of congenital cyanosis as it appears in those cases where oxygen unsaturation has increased up to or beyond its threshold value (6.5 per cent), either through direct admixture of venous with the arterial stream, as in absence of the cardiac or arterial septa or transposition of the great trunks (Lundsgaard's α factor), or increased oxygen consumption in the capillaries, as in the right-sided valvular lesions (D factor).

Two causes militate directly to the distress of such patients: (*a*) the inadequate supply of oxygen to the tissues; (*b*) the pressure-effects upon these of abnormal fullness in the venous reservoir resulting from the overfilled right heart such as exists both in the right-sided valvular lesions and in large septal defects. In addition to the objective phenomena of oxygen-unsaturation, cyanosis, clubbing, cyanosis retinæ, and polycythemia, the special subjective symptoms that call for relief are: palpitation and dyspneic attacks with transient increase of cyanosis, chilliness of the surface and extremities and such evidences of venous engorgement as insomnia and epistaxis. Treatment is, of course, entirely palliative in this unfortunate group of cases, *preventive* in the sense only of retarding the downward course of the disease by strengthening the power of the heart muscle within its limitations, and *symptomatic* only in so far as the distressing symptoms of oxygen unsaturation or venous plethora admit of alleviation.

Symptomatic Treatment of the Cyanotic Group.—The palliative measures available may be enumerated as follows:

Bloodletting may be required to assist in depleting an overloaded right ventricle. This may be carried out by venesection, 15 or 20 ounces being withdrawn, often to the great relief of the patient, or by the application of two or three leeches over the liver; the latter method is preferable in children, but care should be taken to avoid alarming very young subjects.

Various *medicinal remedies* may be employed. Stimulants for the relief of the dyspneic attacks, and the temporary support of the failing myocardium, in childhood the most suitable being small doses of brandy or ammonia and ether, caffein citrate, adrenalin, etc., or such antispasmodics as cannabis indica may be tried. To allay the nervous irritability and cough, so troublesome in the later stages, sedatives or narcotics such as the bromids for more prolonged dosage, or small doses of heroin (gr. 1/12) or morphia gr. 1/8 with atropin gr. 1/100. As hypnotics for the insomnia that is often a distressing feature, veronal gr. vii ss, trional gr. x or sulphonal gr. xx (adult doses) may be used, and for more intractable cases a chloral mixture.

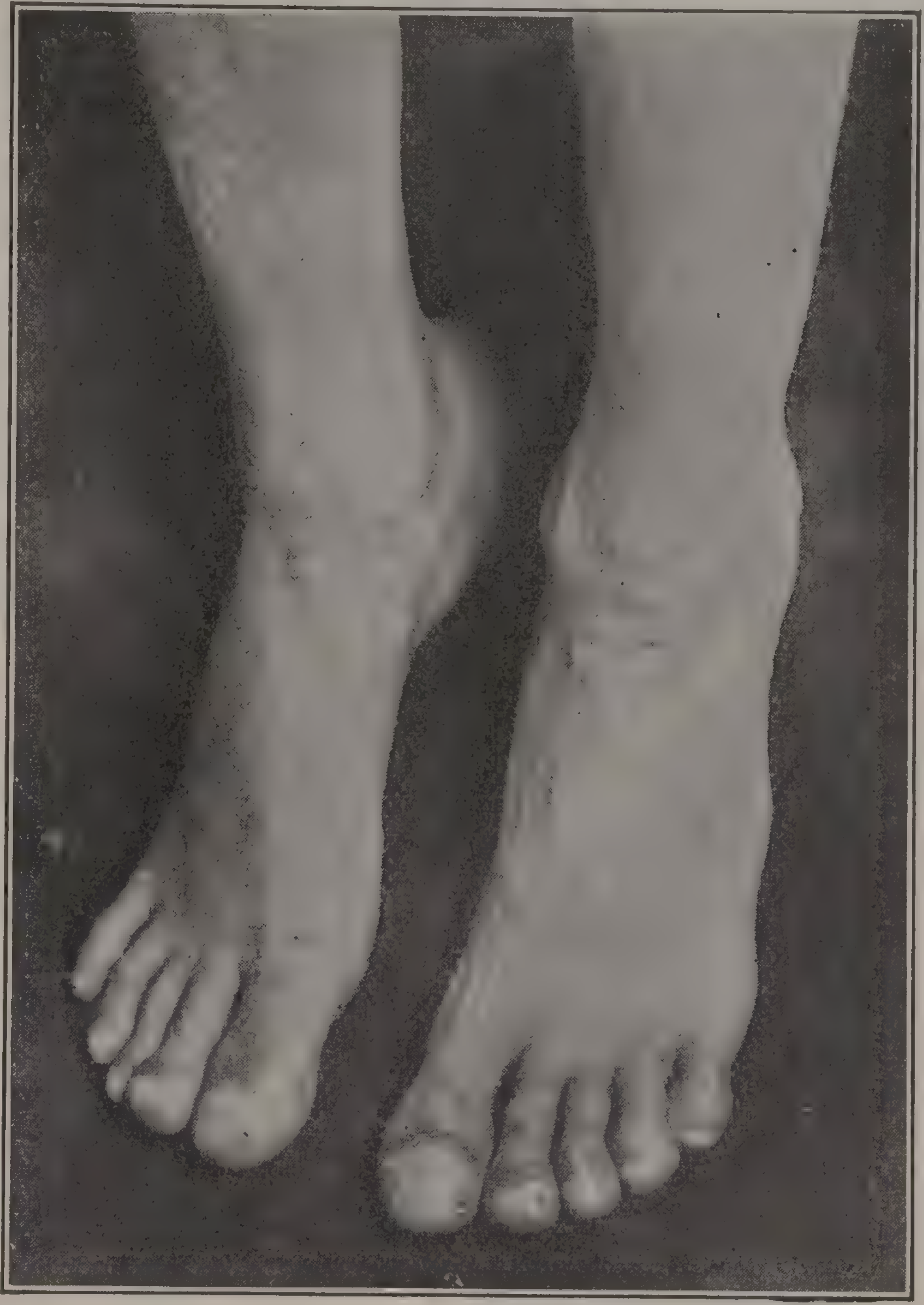


FIG. 10.—CLUBBING OF TOES IN CONGENITAL CYANOSIS. (Courtesy of Dr. John Howland, Baltimore.)

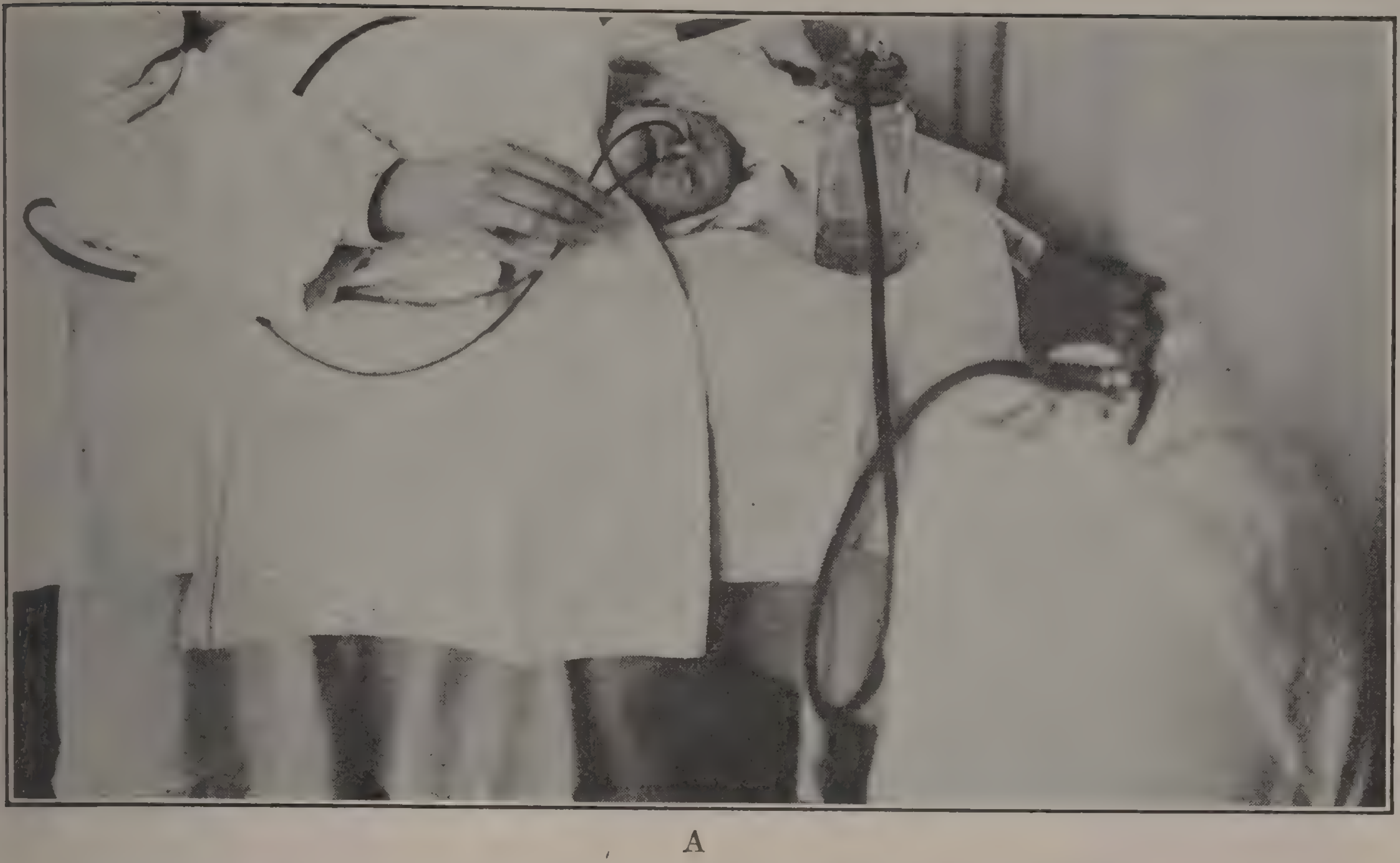
Treatment by Inhalation of Oxygen.—This treatment, though entirely futile in true *morbus cœruleus* for reasons given below, must be retained in the therapeutic armamentarium, both because this is an important diagnostic measure in cardiac defects, and because it may be of actual assistance in cases where certain complicating pulmonary conditions contribute to the cyanosis, although not its essential cause. The dramatic relief which follows this measure in cyanosis due to deficient pulmonary oxygenation, as in fetal atelectasis (Lundsgaard and Van Slyke's *l* factor), brings the question as to its indication for congenital cyanosis immediately into the foreground. One of the greatest services rendered by Lundsgaard and Van Slyke in this field is the clear demonstration of the reasons why oxygen inhalation brings absolutely no reduction of the unsaturation in congenital cardiac disease proper; for cyanosis is here, at least in the great majority of cases, the result either of a "right to left shunt" of venous into arterial blood through a septal defect (alpha factor —see Fig. 3) or of an increased oxygen consumption in the tissues from peripheral stasis (D factor), as in right-sided valvular lesions; and therefore oxygen administered through the mechanism of the pulmonary circulation to blood already fully saturated in the lungs of course cannot mitigate the effect of the unsaturated blood entering from an entirely extraneous source.

On these grounds oxygen administered by the pulmonary route is to be definitely recognized as of no direct therapeutic value in congenital cyanosis. Its rapid action, however, in cyanosis of pulmonary origin, makes it of much use in the diagnosis or elimination of this possibility, and, where the etiology of the cyanosis is doubtful, especially in young infants in whom atelectasis may be at least a contributing cause, it should be used both to assist in establishing the actual condition present and also for its possible remedial effect in suitable cases.

The old methods of administration by funnel or otherwise are futile; Haldane has demonstrated that 98 per cent of the gas is lost in giving it in this way (or, to use Meltzer's terms, the air is not enriched by the funnel method more than 2 per cent). Oxygen chambers enclosing the entire subject have been devised by Stadie and others, but a practical clinical procedure has not yet been invented; and the tent method, as used by Leonard Hill (in which the upper part of the body is enclosed in a cardboard or other air-tight box, into which oxygen is admitted and inhaled pure) is adapted to emergency use but is not ideal. For adults and older children, the most practical apparatus to date is along the lines of that described and figured by Haldane in which the minimum amount of oxygen required to relieve the cyanosis is first estimated (usually 1 to 3 liters per minute) and this delivered into a proper collecting bag and thence administered (through warm water) to the patient by tubing supplied with a mica non-return expiratory valve, face mask and nose clip being duly applied.

Barach and Woodwell substitute a soda-lime mixture permitting of re-breathing and a two-way valve connecting the patient with the air for Haldane's reducing valve, and use an ordinary rubber mouthpiece with nervous patients instead of the mask, omitting also the nose clip, on the ground that a sufficient admixture of oxygen is obtained when the patient is attached to the oxygen tank by combined nose and mouth-breathing. Their apparatus is figured in the "Studies in Oxygen-Therapy" by these authors.

In infants or other subjects incapable of coöperation a Y-shaped tube provided with two small catheters may be inserted in the nostrils, or a catheter (protected by a collar as described by Baikwin) inserted in the mouth and held in place by tapes. The accompanying illustration (Fig. 11) shows the application of the two latter methods.



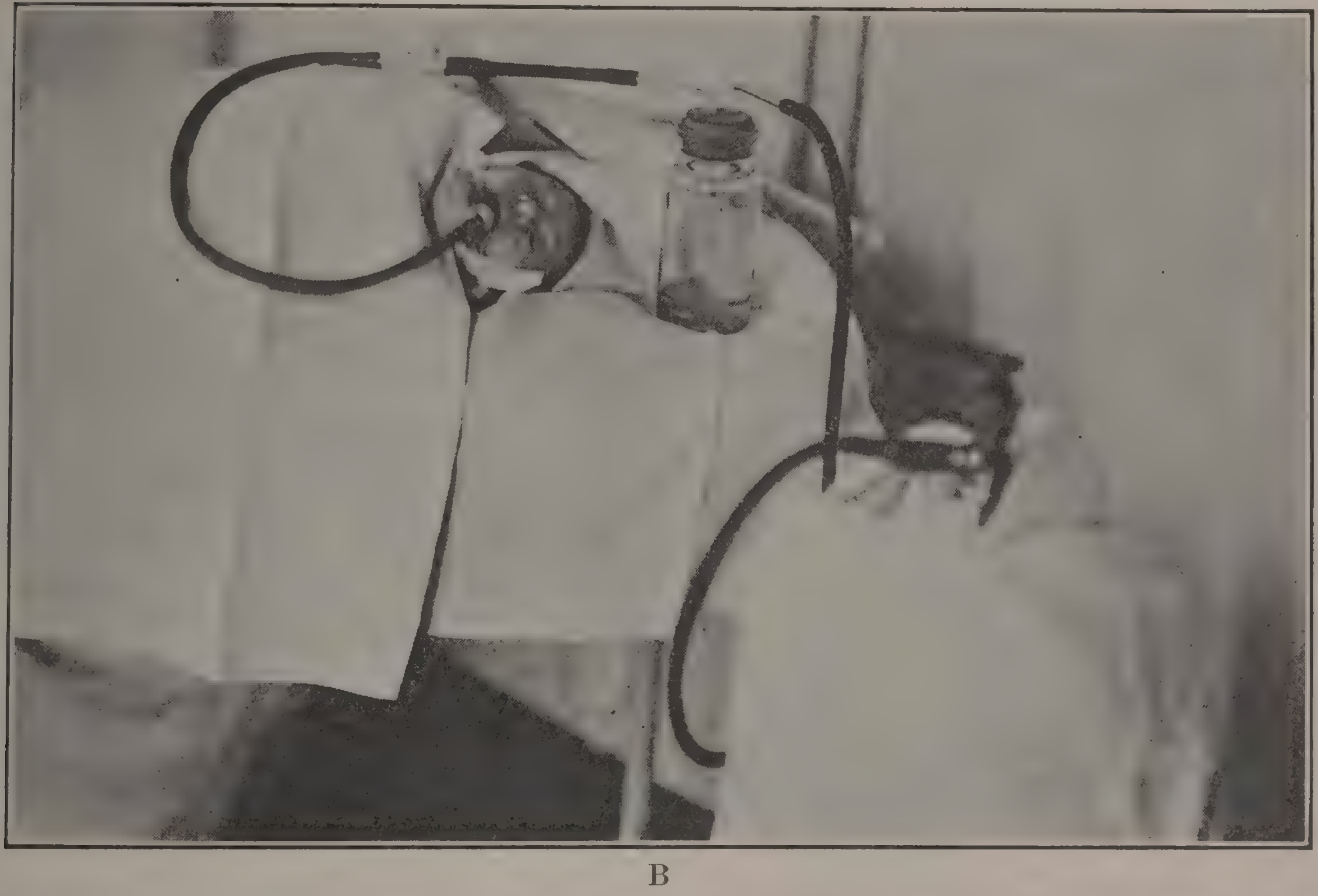


FIG. 11.—METHODS FOR ADMINISTERING OXYGEN TO YOUNG INFANTS. (Courtesy of Dr. Bret Ratner, New York.)

A. Oxygen can be given by means of two fine catheters inserted into the nostrils. The catheters are well lubricated and connected to the oxygen tank by means of a Y-tube.

B. Oxygen administered by means of a rubber tube (1 cm.) inserted into the mouth. The tube can be held or kept in place by means of adhesive plaster or a tape run around the child's head

Intra-vascular Oxygen Administration.—This subject of oxygen therapy in congenital cyanosis cannot be dismissed without reference to the recent experimental work (as yet unpublished), carried out by Dr. E. T. Lumsden at the Lister Institute, by which he has demonstrated that pure oxygen may be administered intra-arterially to rabbits, cats and dogs, under due precautions as to quantity and rate, without the production of clotting or gas embolism. Of still greater practical interest are the results reported by Galata, who has injected oxygen intravenously into normal dogs at a maximal dose of 2 c.c. per kg. per minute, without untoward effects; in the case of prolonged injections it was found advisable not to go beyond 1.5 c.c. per kg. per minute, which is an amount that would be sufficient to relieve an extreme degree of oxygen unsaturation. Intravenous doses of 3 c.c. per kg. could be administered for a few minutes only. The maximal innocuous dose remained the same whether the veins for the injection were near to or far from the heart, and even if the animal was in an asphyxiated condition. These investigations have not yet been extended to human subjects but it is readily seen that if, in the course of such researches, it be found possible to establish a method by which oxygen may be introduced with safety directly into the circulation over even a short period, the relief to the patient from his extreme degree of oxygen unsaturation might last long beyond the time of administration, and thus secure to these distressing cases the same alleviation that is derived from oxygen inhalation in anoxemia of pulmonary origin.

Preventive Treatment of the Cyanotic Group.—Under this heading are considered those general hygienic measures that may retard, even though they cannot finally arrest, the downward trend of the disease. In this connection the following judicious and valuable statement has been kindly supplied to the writer for the purposes of this article by Dr. Edith Bronson of San Francisco, a pediatrician whose experience in the physiotherapy of cardiac disease in children makes her an authority on this subject.

"*The Cyanotic Group Which Lives to School Age.*—From birth the child should be under the most careful supervision in respect to diet and hygiene. Prevention of infections, cold, bronchitis and the diseases of childhood is most important. An infection, which might be of little significance in the ordinary child, may be fatal in one of these children. Each cold should be treated as a serious thing.

"*Character-building.*—Self-control and obedience are most necessary in this type of child. The temptation is, because the child becomes cyanosed on crying, to let him have his own way in everything. This is entirely wrong. He must learn self-restraint if he is to live, and he learns it with least injury to himself in infancy. Even with the risk of convulsive attacks, the lesser danger is not to "give in" to the infant or young child, and his training in these respects is as important as the prevention of infections, and much more difficult. It can be satisfactory only when the mother is moderately intelligent and has complete confidence in the physician. As a child usually can be taught personal cleanliness, so he can be taught exercise habits which correspond to his cardiac tolerance. Instead of being constantly nagged by the anxious mother, great pains should be given to teaching him games which he may play. He should not be made an invalid, sitting about and allowed no exercise. The standardization of games in regard to the work they require may be useful. Personally, I am more inclined to think that the education of the individual child to keep within his own tolerance limits is more important. This is easier in children with congenital hearts than in those suffering from acquired disease, because the effect of overwork is immediate, and more or less distressing. The average intelligent child of five years knows when he is out of breath, and if he has been properly brought up, can be taught to keep his play exertion below the tolerance limit. When the school age arrives difficulty in control is increased. Competitive games must be eliminated and in others the child is tempted to do his utmost. Many who have progressed fairly satisfactorily in the preschool age show constant cyanosis, and extreme cyanosis on exertion, after a year or more of school life. (This should be more frequently remembered in differential diagnosis between congenital and acquired heart disease in young children.) At this age and throughout life the benefit of the early training in self-control appears.

"*The question of institutional treatment* of this much handicapped group must be noted. A special school is of course essential, also play supervision. The advantage is the elimination of competition, and a lessened liability to intercurrent infections. The disadvantages are many. Ultimately the individual, if he lives, must return home and face his life without the protection which an institution affords. With the possible exception of intolerable home surroundings, which no amount of social work can modify, it seems wiser that he grow up himself in the environment in which he must live.

"*Tests of Cardiac Function.*—In the cyanotic group set tests give little added information. I have used the ability to recover after extreme dyspnea. My method is somewhat as follows: Rest (prone) until respiration, pulse and systolic pressure are constant. Exercise—as hopping, jumping, climbing stairs, or calisthenics—until the child is "tired out." Repeat (child lying down quickly), respiration, pulse and systolic pressure. Repeat in one minute, again in five minutes, and longer if the return to normal has not occurred. In the normal athletic child the return to normal, even after striking dyspnea, is within two minutes. In fact the pulse usually slows below the starting point within two minutes. The respiratory rate is the latest to reach normal. The type of systolic pressure curve is characteristic, and has been described in detail by Barringer of New York; also the type of delayed rise and prolonged fall which goes with cardiac inefficiency. (I am coming to believe that the recovery from dyspnea is just as accurate a gage as the more difficult to obtain blood-pressure curve.)"

"Now in using the above routine, I have found certain instances of low systolic pressure which actually seemed to be decreased with exertion. Example—Female, 7 to 8 years, undersized, deeply florid lips and slightly clubbed fingers, seen first about two years ago, when I was especially interested in developing my physiotherapy technic. I put her in the University of California Hospital, for physio-therapy treatment. Her systolic pressure was 76.9 and obtained no rise with exercise, in fact, as dyspnea and cyanosis increased, no sound came through by the auscultatory method. Distress was great even after hopping on one foot only 15 to 20 times. Recovery was rapid when put back to rest, and the systolic pressure returned to her normal. Graded exercise over a period of weeks gave no improvement in these signs. She lost weight in the hospital. This child I have seen at least once a month for two years. She had very diseased tonsils with recurring acute attacks. For about a year the mother would not take the risk of tonsillectomy. For the past year since tonsillectomy, she has improved strikingly, gained 11 to 12 pounds, has a systolic pressure of 86 to 88. She attends school and within her limits is a normal child. Her exercise tolerance is greater, but still very limited. She has so far remained free from intercurrent infections. Life in this instance has been made happier by removal of a handicap in the shape of recurring illness, and by a wisely regulated régime, of which daily periods of open-air exercise are an established part."

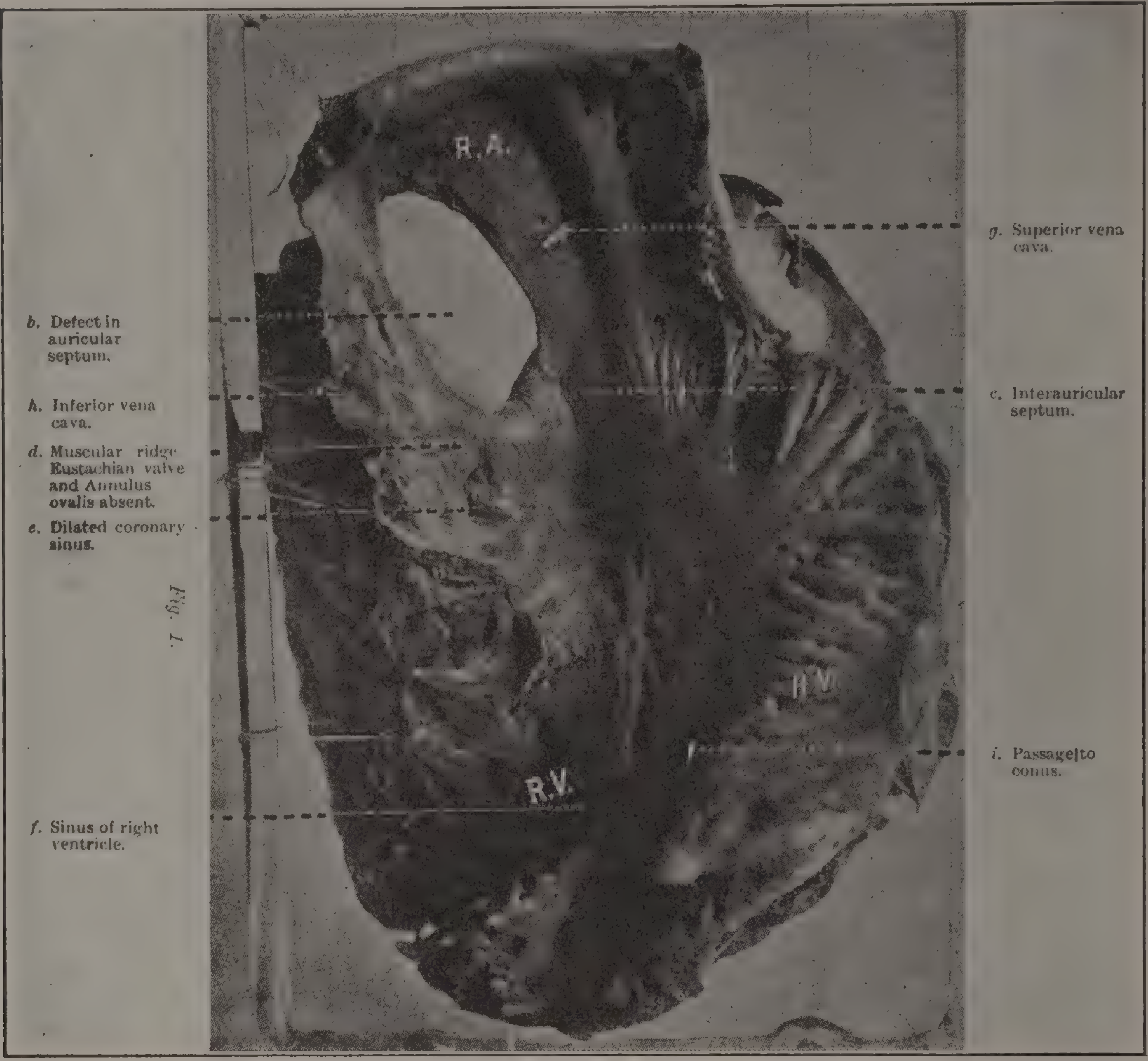
Treatment of the So-called Non-cyanotic Group

In the clinical classification outlined on page 332 (which, it is interesting to note, is the same as that laid down by Bamberger in 1857), two sets of "non-cyanotic" cases, both of clinical significance, are distinguished: (*a*) those presenting abnormal communications between the venous and arterial streams, to be enumerated as localized defects of the interauricular, interventricular, or aortic septa, and patent ductus arteriosus, which usually produce distinctive physical signs but no symptomatology until a pathological rise of pressure in the pulmonary circulation supervenes, producing a reversal of flow and an influx of venous into arterial blood through the defect and a resultant "late" cyanosis follows; (*b*) cases which present no such abnormal opening but only certain minor malformations (such as bicuspid valves, anomalous septa, etc.) producing irregularities of the blood current that may be the source of cardiac strain or supply a *locus minor resistentiæ* to infection, but that do not lead to the phenomena of deficient aëration.

Of these two sets of cases, the former, namely the cardiac septal defects, is naturally the more serious, for to the dangers common to both are added those incident to a terminal oxygen unsaturation. From the standpoint of therapeusis, the preventive treatment of these cases is of much interest, and is of infinitely greater social importance than is the problem of the merely palliative treatment of true *morbus cœruleus* discussed above. For the majority of these patients begin life as physiologically normal individuals with a healthy metabolism and respiratory exchange, and with a good expectation of life, provided they can be safely piloted through the special difficulties and dangers incident to the presence of the defect.

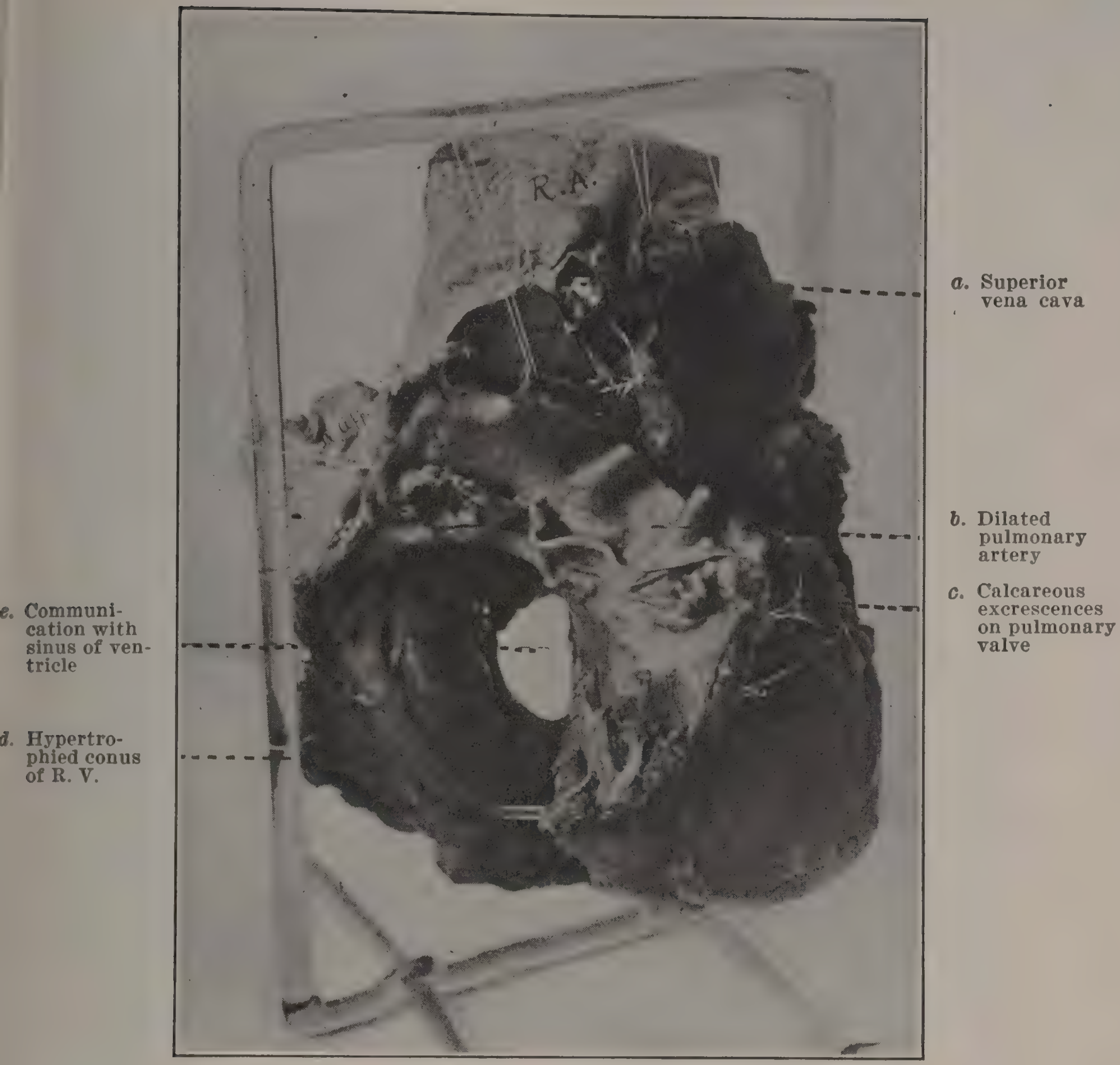
During the last decade, the science of cardiology in all its details has advanced so far that an exact diagnosis of most of these cases, at least of localized ventricular septal defects and patent ductus arteriosus, is usually made in our larger hospitals and cardiac clinics, and is entered as such upon their records. It is therefore interesting to note that no special mention or place is as yet accorded them in the classification by Dr. T. Stuart Hart now in general use by the Associated Cardiac Clinics and the New York Association for the Prevention and Relief of Heart Disease, where they are apparently relegated from their loud atypical murmurs to the "possible" organic lesions (Class IV) until a diagnosis is made. The opinion is submitted here that, once diagnosed, these localized septal defects during their "compensated" stage, are not only both organic lesions presenting no symptoms (Class I), but are also members of that most important "Potential" group (Class V), as presenting an overloaded pulmonary circulation which in no way embarrasses the patient at the time of observation, and as providing a seat of strain or impact in the endarterium or endocardium that may some day form a nidus for the invasion of virulent microörganisms circulating in the blood-stream. It is therefore suggested that all cases of "compensated" septal defects, when diagnosed as such, should be definitely recognized under this group, by the early removal of foci of infection, and the prompt treatment of rheumatism by rest, tonics and salicylates; by the determination of the individual's cardiac tolerance; and thereafter by the prescription of exercise or play in accordance with the patient's functional occupation such as will meet his social and personal requirements, while shielding him in so far as this is possible from the peculiar dangers that threaten him through the nature of his organic lesion.

The mechanical conditions in septal defects and the special difficulties under which these cases labor in relation both to overloading of the right heart under the physiological condition of "left to right shunt" (Fig. 7) and the pathological features of late cyanosis introduced by a terminal reversal of flow (Fig. 3) as well as the peril in which these patients stand of intercurrent infections about the margins of the defects, are illustrated by the following specimens in the McGill Medical Museum, which are figured here in order to elucidate and emphasize these important and too often forgotten points.


R.A.
g. Superior vena cava.
b. Defect in auricular septum.
h. Inferior vena cava.
c. Interauricular septum.
d. Muscular ridge Eustachian valve and Annulus ovalis absent.
e. Dilated coronary sinus.
Fig. 1.
R.V.
R.V.
i. Passage to conus.
f. Sinus of right ventricle.

A

Fig. 12 A, Case 1.—To Illustrate Left to Right Shunt with Terminal Reversal of Flow. Large Defect at the Upper Part of the Interauricular Septum (Persistent Ostium Secundum). Hypoplasia of the Aorta and Great Dilatation of the Pulmonary Artery with Marked Thickening and Calcification of the Pulmonary Valves, Hypertrophy and Dilatation of Right Chambers. Interior of right chambers showing large defect at upper part of interauricular septum, absence of Annulus ovalis, and great hypertrophy and dilatation of right chambers. From a woman, aged 64, with left to right shunt of the circulation until six months before death when a reversal of flow occurred with resultant "cyanose tardive" following bronchopneumonia. (From a specimen in the Pathological Museum of McGill University presented by F. W. C. Mohr, reported by M. E. Abbott and J. Kaufman, *Journal of Pathology and Bacteriology*, 1910.)


R.A.
a. Superior vena cava
b. Dilated pulmonary artery
c. Calcareous excrescences on pulmonary valve
e. Communication with sinus of ventricle
d. Hypertrophied conus of R. V.

B

FIG. 12 B, CASE 1.—TO ILLUSTRATE LEFT TO RIGHT SHUNT WITH TERMINAL REVERSAL OF FLOW. LARGE DEFECT AT THE UPPER PART OF THE INTERAURICULAR SEPTUM (PERSISTENT OSTIUM SECUNDUM). HYPERPLASIA OF THE AORTA AND GREAT DILATATION OF THE PULMONARY ARTERY WITH MARKED THICKENING AND CALCIFICATION OF THE PULMONARY VALVES, HYPERTROPHY, AND DILATATION OF RIGHT CHAMBERS. Anterior view of 12 A showing calcareous pulmonary valves and dilated conus of right ventricle. From a woman, aged 64, with left to right shunt of the circulation until six months before death when a reversal of flow occurred with resultant "cyanose tardive" following bronchopneumonia. (From a specimen in the Pathological Museum of McGill University presented by F. W. C. Mohr, reported by M. E. Abbott and J. Kaufman, *Journal of Pathology and Bacteriology*, 1910.)

Case 1 (Fig. 12 A and B), to illustrate the development of "right to left" shunt supervening at the end of a long life of perfect competence in which a "left to right" shunt (Fig. 7) with great overloading of the pulmonary circulation had persisted for over sixty years until a raised pulmonary induced a reversal of flow (Fig. 3). Large defect of the interauricular septum, size of a fifty-cent piece with hypoplasia of the aorta and great dilatation of the pulmonary artery, hypertrophy and dilatation of the right ventricle and marked *thickening and calcification of the pulmonary valves from overwork.* A hard-working charwoman, aged 64, was perfectly healthy, and presented no sign of cyanosis until six months before death, when she became cyanosed and developed bronchopneumonia and terminal cyanosis. Reported by us in *Journal of Pathology and Bacteriology,* 1910.

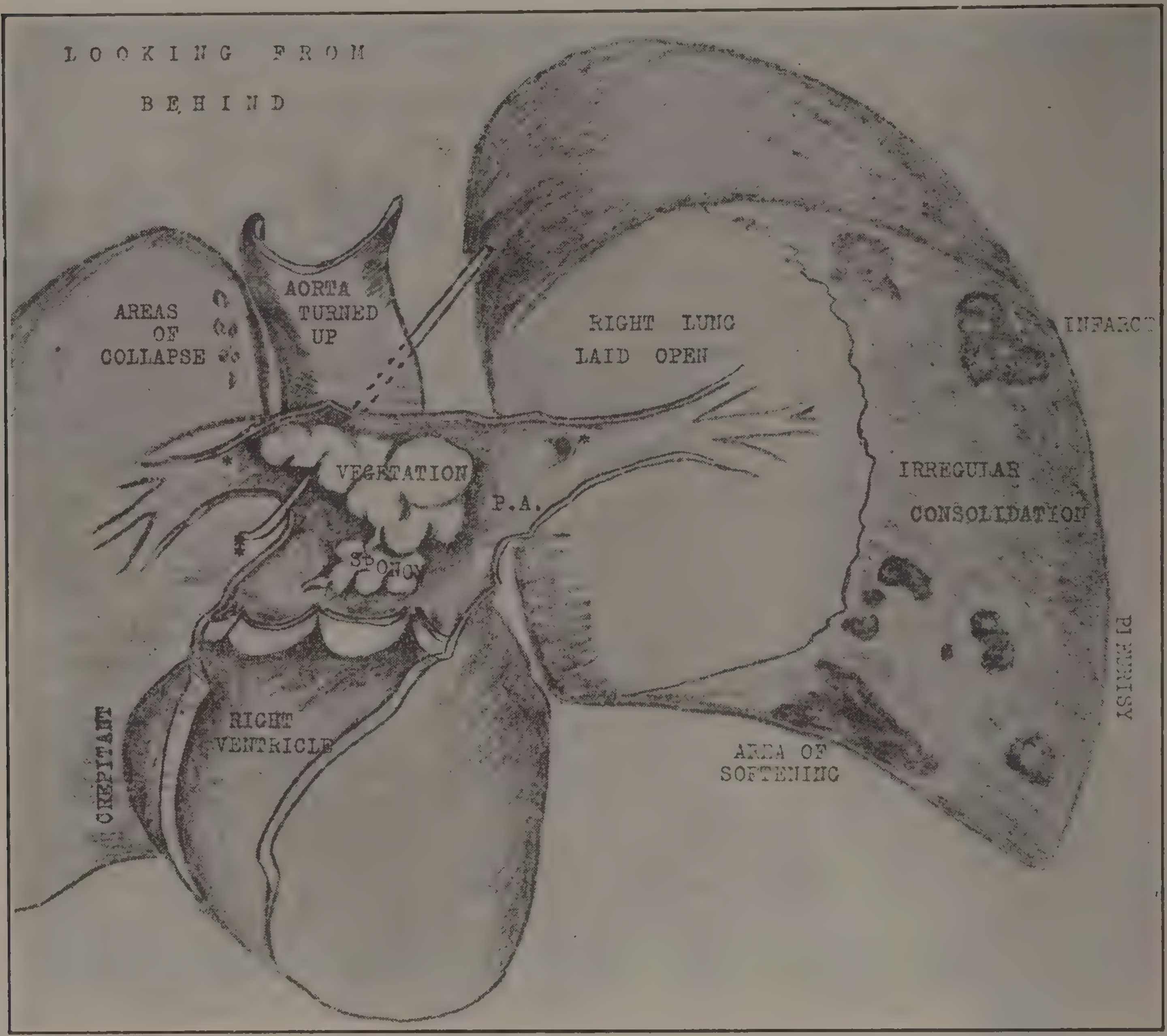
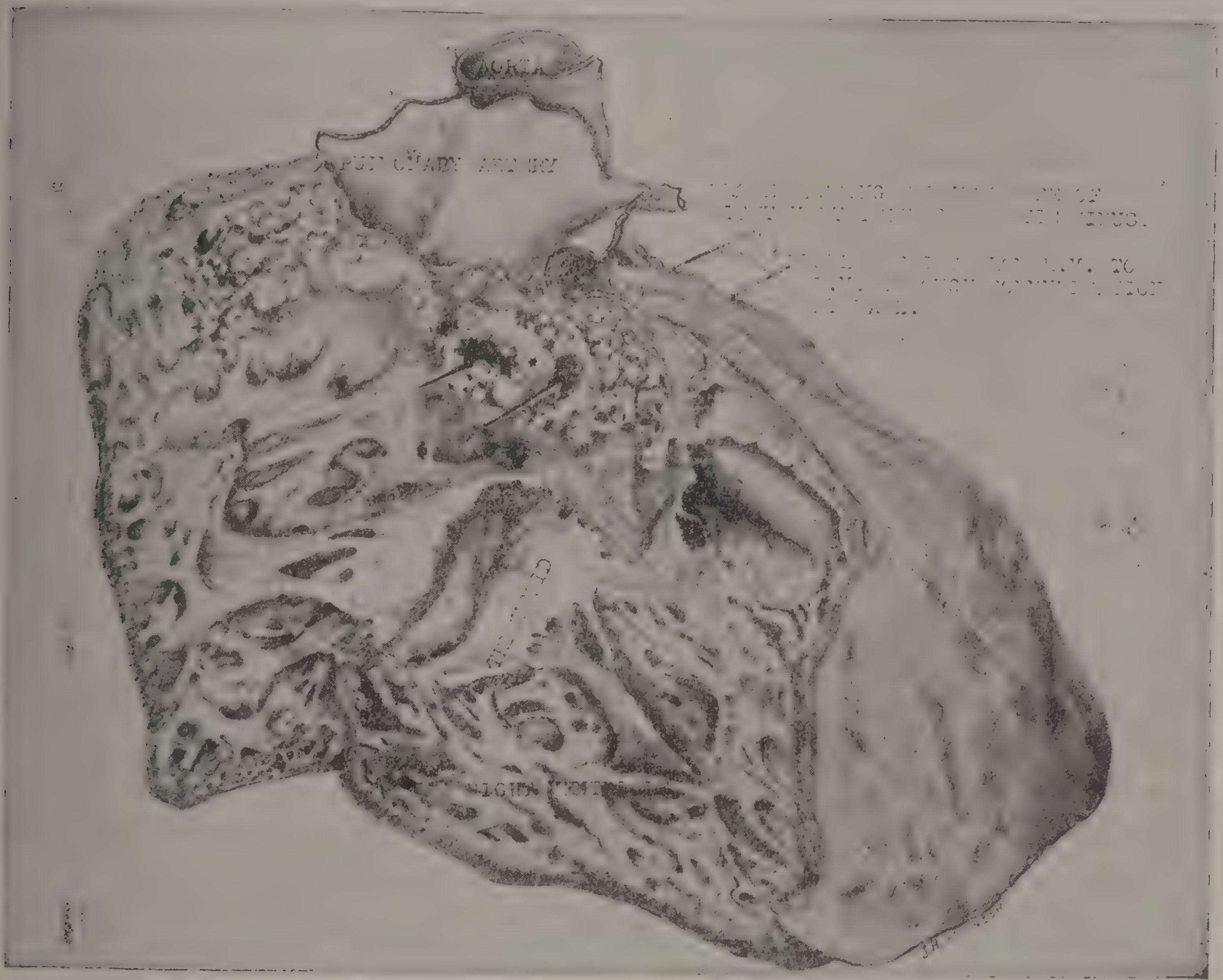


FIG. 13, CASE 2.—PATENT DUCTUS ARTERIOSUS WITH ACUTE INFECTIVE ENDARTERITIS ABOUT PULMONARY ORIFICE OF DUCTUS AND MULTIPLE EMBOLIC ABSCESSES OF LUNG. PNEUMOCOCCUS INFECTION. Note huge thrombotic vegetations blocking ductus and healthy pulmonary valves. (*To illustrate incidence of an infective process on the right side in a case of left to right shunt.*) (From a specimen presented to the Pathological Museum of McGill University by Professor O. C. Gruner. Reported by Drs. W. F. Hamilton and M. E. Abbott, Trans. Assn. Am. Phys., 1914.)

Case 2 (Fig. 13), illustrating the invasion by virulent microörganisms of the margins of the defect in the right ventricle and pulmonary artery in a case of left to right shunt of the blood-stream as shown in Figure 7. Patent ductus arteriosus with huge thrombotic masses of malignant endarteritis about the pulmonary orifice of the ductus, and embolic abscesses of the lungs. From a girl, aged 19, dying of pneumococcus septicemia. Reported in *Transactions American Physicians,* 1914.

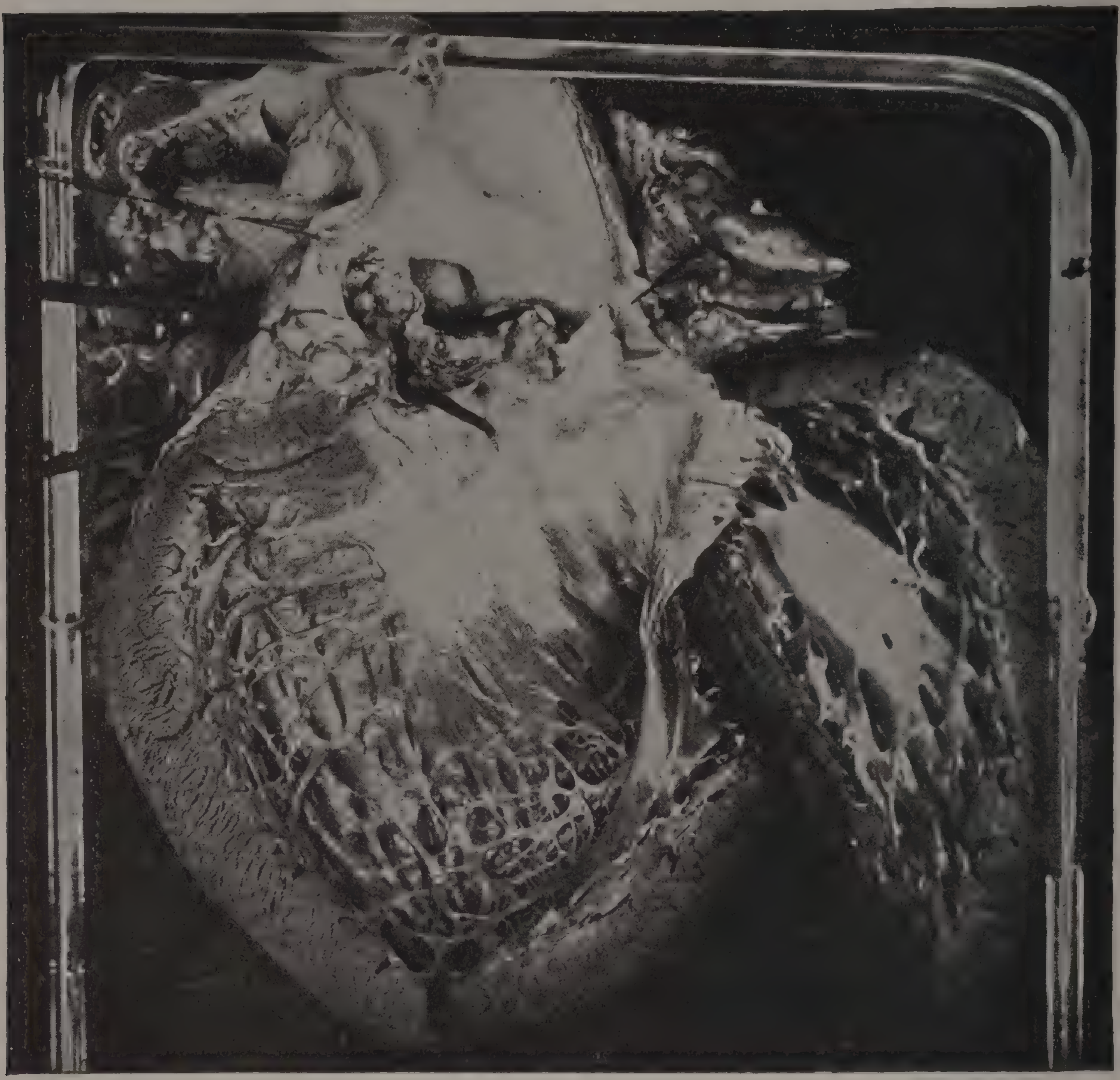


A

Fig. 14.—Aneurysm of the Right Aortic Sinus of Valsalva Rupturing into the Conus of the Right Ventricle with Defect of the Anterior Interventricular Septum and Malignant Endocarditis of the Margins of the Defects in the Right Mural Endocardium of the Right Ventricle Adjacent and Opposite These and of the Pulmonary and Aortic Valves.

A. View of interior of right ventricle, showing the tubular ruptured aneurysm, and the massive vegetations clustered about and opposite to the defects and on the pulmonary valves. (*To illustrate the incidence of an acute infective process in a case of left to right shunt.*) (From a specimen in the Pathological Museum of McGill University. Republished from the *Sir William Osler Anniversary Volume,* 1919, with permission of Paul B. Hoeber, Publisher.)

Case 3 (Fig. 14 A and B, illustrating same point as Case 2). Ruptured congenital aneurysm of right aortic sinus of Valsalva with localized defect of interventricular septum. Malignant endocarditis of conus of right ventricle, margins of defects, pulmonary and aortic valves. From a man aged 38, presenting signs of communications between aorta and base of right ventricle and pneumococcus septicemia. Reported, in *Osler Anniversary volume,* 1919.



B

Fig. 14.—Aneurysm of the Right Aortic Sinus of Valsalva Rupturing into the Conus of the Right Ventricle with Defect of the Anterior Interventricular Septum and Malignant Endocarditis of the Margins of the Defects in the Right Mural Endocardium of the Right Ventricle Adjacent and Opposite These and of the Pulmonary and Aortic Valves.

B. Interior of left ventricle showing the openings of the defects above and below the right thickened and inflamed right aortic cusp. (*To illustrate the incidence of an acute infective process in a case of left to right shunt.*) (From a specimen in the Pathological Museum of McGill University. Republished from the *Sir William Osler Anniversary Volume,* 1919, with permission of Paul B. Hoeber, Publisher.)

From the above it is evident that for the adequate treatment of septal defects preliminary knowledge is necessary, first as to the existence of the defect itself which must be diagnosed before the case can be transferred from the "possible" to the "potential" class; for not all loud murmurs of irregular localization and occurring in young subjects are other than functional; and secondly as to the "cardiac tolerance" of the individual which must be estimated both in order that he may be permitted to take his part in life to the extent of his capacity, and that the physician may judge of the degree of cardiac strain he is supporting without present embarrassment. Both these essentials require brief discussion here.

Special Diagnostic Methods.—In a fair proportion of cases, the diagnosis can be readily established by the characteristic physical signs that exist, whether a gaping foramen ovale with its concomitant hypoplasia of the aorta, a localized ventricular septal defect, a patent ductus, or the roaring continuous thrill and murmur of a congenital communication between the two circulations above the aortic valves (defect of aortic septum); and such articles as those by Le Houx on "Maladie de Roger"; "Patent Ductus Arteriosus," by Gibson, Goodman, Hamilton and Abbott (Fig. 13), Wells, and Stoddard; "Two cases of Patent Foramen Ovale," and "Ruptured Aneurysm of the Right Aortic Sinus of Valsalva," by Abbott, and "A case of Persistent Ostium Primum," by Abbott and Kaufmann (Fig. 12), should be consulted as well as the various monographs mentioned above. In others, however, the condition is latent or presents confused and indistinctive signs, and in these a method for the determination of the presence or absence of a functional shunt of the circulation is highly desirable, as also for greater exactitude in all. As indicated above a right to left (alpha) shunt is not only recognizable by its effects, but it can readily be determined and quantitatively calculated (Lundsgaard and Van Slyke). The more difficult and far more important problem from the clinical standpoint of recognizing *the presence of the left to right shunt* in compensated septal defects, has not yet been satisfactorily solved, though attempts have been made by Plesch by determination of the oxygen content in the pulmonary circulation and by Boothby and Abbott of its "basal carrying tension" for carbon dioxid (which should vary inversely as the volume).

This problem is in itself a simple one, namely, the determination of the increased volume of blood that exists in these cases in the pulmonary circulation, which should be obtained by calculation of the minute-volumes from the difference between the arterial and true venous oxygen contents. The technical difficulty lies in obtaining the latter, for the great variation in oxygen saturation in the different veins of the body makes the blood from these channels useless as an exact index of that in the right heart. Up to the present time therefore the only way of ascertaining the venous oxygen content of the pulmonary circulation appears to be to determine that of the alveolar air by the nitrous oxid method by means of the Krogh spirometer,[1] and, in the hands of workers versed in this technic, it is hoped an early solution may be achieved. An advance in laboratory methods that will enable the physician to determine a left to right functional shunt in a patient who presents no distinctive physical sign of the strain and danger under which his circulation labors will be of value in the preventive therapy of the non-cyanotic group, commensurate only with the relief that will follow the intra-arterial administration of oxygen in the cyanotic cases, when the latter comes to be feasible as a practical clinical procedure.

As in so many other ways in the domain of cardiac pathology a brighter future may thus be about to dawn even upon the clouded horizon of congenital cardiac disease!

Estimation of Cardiac Tolerance.—When one glances at the condition figured in Figure 12 and observes the hidden factors of cardiac strain that are at work in such cases as this, and further notes, in Figures 13 and 14, the visible ravages of the malignant enemy that hovers over the unfortunate subjects of cardiac defects, one realizes the great importance to the physician of a knowledge of the functional efficiency of the myocardium in these patients. This must ever remain only an approximate estimate, and the best criterion is always gained from skilled personal observation of the individual's "reaction" to work and play. Nevertheless the exact knowledge of normal values, and the standardization of "exercise tolerance" and "energy-index tests" that have resulted from the organized researches of the past few years have certainly yielded valuable aids to the physician's use which are both easy of application and sufficiently reliable to be of use. Such methods depend upon the effects of a graded amount of exercise or work upon the pulse and respiration rate, height of systolic pressure, pulmonary ventilation, vital capacity, etc., both as regards the amount of increase, time interval before its production, and duration of the reaction. The exercises used are mostly hopping, stair-climbing, running, dumb-bell swinging or other gymnastics, or such tests as changing from the erect to the recumbent posture, raising a given number of foot-pounds, etc. Full details of their application may be obtained from several current articles, notably those by Norman and Herrick. The British Medical Research Committee's Report on D. A. H. also contains much of interest in this connection. Chiefly through the researches of Peabody, changes in vital capacity are recognized as one of the simplest and probably the most reliable index of myocardial efficiency. Normal values have been established by him as 4,000 to 5,000 c.c. in men and 3,000 to 4,000 c.c. in women, the amount varying with age, height, weight, surface area, and occupation of the individual. Elaborate studies into the various factors influencing the normal vital capacity of children of different types, ages, classes and races and the relative values obtained by different forms of spirometer have been made by May G. Wilson and D. J. Edwards. The best instrument for general use is conceded to be

---

[1] Those interested in this technic will be pleased to know that an improved model of Professor Krogh's spirometer is now available for use through H. N. Elmer of Chicago, who is his agent for this continent.

that made by W. E. Collins of Boston, at the direction of Dr. F. W. Peabody.

The following "Exercise Tolerance Test" carried out at the Children's Memorial Hospital, Montreal, in the Cardiac Clinic of Dr. H. P. Wright as a routine measure in cases, is sufficiently simple to act as an illustration of such procedures. The child's weight, height, temperature, pulse, blood-pressure and vital capacity are taken; he is then made to run along a corridor some 20 yards long and up a stair some 20 feet high, and back without stopping; one minute later the pulse and vital capacity are again taken and five minutes after the systolic blood-pressure (which falls last). In a perfectly efficient heart all three values should have returned to normal after these intervals, the exercise itself producing an increase of some 4 to 6 beats in the pulse, 100 to 200 c.c. in vital capacity, and 5 to 20 mm. in systolic pressure. This estimate is repeated once weekly.

The diagnosis having been made and the degree of exercise tolerance determined, it remains to establish for these "non-cyanotic" cases of "compensated" cardiac septal defect a protective hygienic regimen such as is prescribed for all members of the so-called "potential" group, and that is especially indicated at the periods of puberty and adolescence in order that at this time of added strain, a "non-cyanotic" case may not pass over into the cyanotic group. A preventive treatment along similar, though less severe, lines to that laid down above by Dr. Edith Bronson for "cyanotic children reaching school age" will be indicated, particular regard being paid to the avoidance of recurrent rheumatic infection by free salicylate treatment, and to the education of the myocardium within its limitations by a judicious physiotherapy, in which special attention is to be given to posture and breathing exercises. In this connection the reader is again directed to the admirable exposition by Dr. Edith Bronson of the graded system of physiotherapy applied under her direction by trained technicians in successive stages to cases convalescing from rheumatic infections, with whom we may rightly class this group of "non-cyanotic" cardiac defects.

Surgical Treatment

Very little is to be said under this heading, but certain recent work of great promise cannot be passed over without mention, as well as the important advance that has been made in the prevention of rheumatism and other infections involving risk of prenatal disease and of later complications, by *early tonsillectomy*. This latter operation, which, fifteen years ago, was rarely carried out under the age of twelve, is now done in childhood with strikingly beneficial results, not only in preventing recurrent attacks of rheumatism, but also, as shown by the figures of E. H. Place in aborting the course of scarlet fever and diphtheria. Goerke is against too early tonsillectomy and advises waiting until the eighth to tenth year, when normal involution has begun.

Tonsillar treatment by radium, replacing the more radical step of removal, is practiced also in many institutions, but it is too soon to pronounce upon results. The subject is reviewed by F. H. Williams. *Early removal of carious teeth* is another important preventive measure that may be classed among surgical procedures.

The most interesting work that has been done on *intracardiac surgery* is that for the *operative relief of mitral stenosis,* a procedure suggested by Sir Lauder Brunton as long ago as 1902. Allen and Graham invented an endocardioscope containing a screened knife which they inserted into the left auricular appendage of dogs, by means of which the various finer structures of the interior of the heart were clearly seen and identified, and an adequate opening made by incision through the tendinous screen of a stenosed mitral valve, the chordæ tendineæ, or other parts as required, under the guidance of the eye. This operation is conducted without hemorrhage or necessity for undue haste, and is well tolerated by the heart of the dog. Recently Cutler and Levine, after several years of animal experimentation, have ventured upon operative interference in a carefully selected human subject, with complete success. The patient, a bright little girl of twelve, presented an advanced grade of mitral stenosis and was suffering from marked embarrassment of the right heart and pulmonary circulation, with hemoptysis and orthopnea, etc. Under a careful technic the pericardium was laid open by a median sternotomy. The heart was freed from its moorings below and reverted to expose the posterior surface of the left ventricle which was entered near the apex by a valvulotome pushed upward to the stiffened and narrowed mitral orifice, both corners of which were incised. There was no bleeding and, after two critical postoperative days, the child made an excellent recovery with marked relief of symptoms, but an extraordinary precordial bulging.

The advanced stage to which this new operative procedure has been brought by animal experimentation and now by successful operation on the human subject warrants one in hoping also that, with the improved technic now possible, the attempt made some years since by Tuffier and Carrel to relieve (inflammatory) pulmonary stenosis by incision of the valvular disc may yet be attended with a measure of success.

Ligature of a patent ductus arteriosus seems feasible, but the mechanical difficulties are great, and we know of no attempt so far in the human subject.

Intravenous and intra-arterial administration of oxygen, as mentioned above, are under experimental investigation and hold out hope of relief from some of the worst sufferings from oxygen unsaturation.

REFERENCES

Abbott, Maude E. Aneurysm of the Right Anterior Aortic Sinus of Valsalva, Contributions to Med. & Biolog. Research, July 12, 1919.

——— Congenital Cardiac Disease, Osler & McCrae's System of Medicine, 2d. ed., iv, 323, 1915.

——— Two Cases of Patent Foramen Ovale, Bull. Internat. Ass. Med. Mus. No. 5, 1915.

Abbott, M. E., and Kaufman, J. Report of a Case of Persistent Ostium Secundum, with, for comparison, a Case of Persistent Ostium Primum, Journ. Path. & Bacteriol., xiv, 1910.

Albasanz. Lesions congénitales du coeur et hérédité tuberculeuse, Rev. españ. de Med., April, 1920; Synop., Arch. de mal. du coeur, March, 1922.

Allen, Duff S., and Graham, Edwarts, A. Intracardiac Surgery, Journ. Am. Med. Ass., lxxix, 1028, 1922.

Baikwin. Oxygen Therapy in Premature Infants with Cyanosis. Arch. Pediat., xxxix, 677, 1923.

Bamberger, H. Lehrbuch der Krankheiten des Herzens, 383, Wien., 1857.

Barach, A. L., and Woodwell, Margaret. Studies in Oxygen Therapy Arch. Int. Med., xxviii, 374, 1921.

Bard and Curtillet. Contribution à l'étude de la physiologie de la maladie bleue: forme tardive de cette affection, Rév. de méd., Paris, ix, 993, 1889.

Blumer, George. Subacute Bacterial Endocarditis, Medicine, iii, 105-170, 1923.

Boothby, W. M., and Abbott, M. E. On the Difference between the Carbon-Dioxide Tensions of the Arterial and Venous Blood as a Diagnostic Sign in Cardiac Septal Defects, Bull. vi, Internat. Ass. Med. Mus., 122, 1916.

Breed, Wm. B., and White, Paul D. Heart Disease in Pregnancy, Boston Med. & Surg. Journ., 188, 94, 1923.

Bronson, Edith. Physiotherapy in the Treatment of the Child with Rheumatic Fever, Calif. State Journ. Med., xx, 357, 1922.

Carpenter. Congenital Morbus Cordis, Rep. Brit. Soc. Dis. Child., vi, 241, 1906.

Cutler, E. C., and Levine, S. A. Cardiotomy and Valvulotomy for Mitral Stenosis. Experimental Observations and Clinical Notes Concerning an Operated Case with Recovery, Boston Med. & Surg. Journ., clxxxviii, 1623, 1923.

Dupré, E. Communication congénitale des deux coeurs par inocclusion du septum interventriculaire. Première observation de la lésion reconnue pendant la vie et verifié après la mort, Bull. Soc. anat. de Paris, v, 404, 1891.

Edwards, D. J., and Wilson, May G. An analysis of Some of the Factors of Variability in the Vital Capacity Measurements of Children, Arch. Int. Med., xxx, 639, 1922.

——— Journ. Am. Med. Ass., vii, 1102, 1922.

Eisenmenger. Die angeborener Defeckte der Kammerscheidewände des Herzens, Ztschr. f. klin. Med., xxxii (Supp. I), 1, 1897.

Fortmann. Zur praktischen Diagnostik angeborener Herzfehler. Deutsche med. Wchnschr., xciv, 71, 1918.

Galata, G. Richerche sulle iniezione endovenose di O_2 vel cane. Arch. di fisiol, xxi, 331, 1923.

——— Abs. in Physiol. Abstrs., ix, 15, London, 1924.

Gibson. Patent Ductus Arteriosus, Med. Press & Circ., I, 572, 1906.

Goerke. Med. klin. Wchnschr., 1, 1749, 1922. Quoted by P. D. White.

Goodman. Patent Ductus Arteriosus, Univ. Penn. Med. Bull., cciii, 509, 1910.

Haldane, J. S. Journ. Path. & Bacteriol., 23, 443, 1920.

——— Respiration, Ch. VII, The Causes of Anoxæmia; Ch. X, Blood Circulation and Breathing, Yale University Press, 1922. (This contains a wealth of references on anoxæmia and blood-gas work.)

Hamilton, Burton E. Notes on the Problem of Heart Disease in Pregnancy, Bost. Med. & Surg. Journ., 987, 1923.

Hamilton and Abbott, M. E. Patent Ductus Arteriosus in Acute Infective Pulmonary Endarteritis, Tr. Ass. Am. Physicians, xxix, 294, 1914.

Harrop. The Oxygen and Carbon Dioxide Content of the Arterial and Venous Blood in Normal Individuals and Those with Anæmia and Heart Disease, Journ. Exper. Med., xxx, 247, 1919.

Hart, T. Stuart. The Functional Activity of the Heart. Method of Classification, Med. Clin. North America, iv, 1312, 1921.

Herrick, W. W. Functional Tests of the Circulation and their Significance, N. Y. State Med. Journ., xxiii, 53, 1923.

Herxheimer, G. Missbildungen des Herzens und der grossen Gefässe. Schwalbe, Morph. d. Missbildungen, iii, iii Teil, iii Lief., 2 Kap.

Hill, L. A. A Simple Oxygen Bed-tent, etc., Journ. Physiol., 55, May 21, 1921.

Hirschfelder. Diseases of the Heart and Aorta, 3d. ed., J. B. Lippincott & Co., 1910.

Keith, Arthur. Malformations of the Bulbus Cordis, Festschrift of the Quatercentenary of Aberdeen University, July, 1906.

L'Abbé, Marcel. Retrécissement mitral pur et nanisme, Presse méd., xvi, 497, 1908.

Lambert, A. Incidence of Acute Rheumatic Fever at Bellevue Hospital, Journ. Am. Med. Ass., 74, 993, 1920.

Le Houx. La cyanose tardive (Maladie de Roger), Thèse de Paris, 1902.

Libman. Characterization of Various Forms of Endocarditis, Journ. Am. Med. Ass., 80, 813, 1923.

——— Brit. Med. Journ., Aug. 28, 1920.

——— Am. Journ. Med. Sc., 625, 1913.

Lundsgaard, C. Studies on Cyanosis, I and II, Journ. Exper. Med., xxx, 259, 1919.

——— Studies on Oxygen in the Venous Blood in a Group of Patients with Circulatory Disturbances, Journ. Exper. Med., xxvii, 179, 1918. Ibid.

——— Untersuchungen ueber das Minutenvolumen des Herzens bei Menschen II. Deutsches Arch. f. klin. Med., cxviii, 513, 1915-16.

Lundsgaard, C. R., and Van Slyke, D. D. Cyanosis, Medicine, ii, 1, 1923.

Mackenzie, Sir James. Heart Disease and Pregnancy, Oxford Press, 1921.

McLelland, J. E., and Ruh, H. D. Syphilis as an Etiologic Factor in Mongolian Idiocy, Journ. Am. Med. Ass., lxviii, 777, 1917.

Meckel. De Cordis conditionibus abnormibus, Dissertion, Halle, 1802.

Norman, T. The Tentative Determination of Cardiac Reserve as a Therapeutic Index, Med. Rev. of Rev., xxvii, 462, 1921.

Parkes, Weber, and Dorner. Lancet, Jan. 21, 1911, quoted by Haldane, Ch. X.

Peabody, F. W. Clinical Studies on the Respiration, Arch. Int. Med., xx, 443, 1917.

Place, E. H. Boston Med. & Surg. Journ., clxxxvii, 434, 1922. Quoted by P. D. White, Progress in Cardiovascular Disease, Ibid., clxxxviii, 674, 1923.

Plesch. Zur Diagnose der kongenitalen Vitien, Berl. klin. Wchnschr., xlvi, 390, 1909.

Poynton and Paine. Researches on Rheumatism, 389, 1913. Quoted by Goodhart and Still, 11th ed., 768, 1921.

Queyrat, Gandry, and Deginguand. Congenital Mitral Stenosis and Dilatation of the Pulmonary Artery—Apparently Congenital Syphilitic in Origin, Bull. et mém., Soc. méd. d. hôp. de Paris, April 29, 1921; Synop., Arch. d. mal du coeur, March, 1922.

Reports of New York Association for Prevention and Relief of Heart Disease, 1915-1922, 325 East 57th Street, New York.

Roger. Arch. gén. de méd., vii, 121, 1880. Quoted by Le Houx.

Rokitansky. Die Defeckte der Scheidewände des Herzens, Wien, 1875.

Simmons. Congenital heart malformations, Intercolon. Med. Journ. Australia, xi, 65, 1906.

Smith, Bellingham. Congenital Heart Disease and its Prognosis, Practitioner, cxix, 365, 1922.

Steffano, S. de. Congenital Cardiopathies and Syphilis, Pediatria, 992, 1920.

——— Arch. d. mal. du coeur, March, 1922.

Stevens, J. A. Mongolian Idiocy and Syphilis, Journ. Am. Med. Ass., xiv, 1636, 1915.

Stockard, C. R. Developmental Rate and Structural Expansion, an Experimental Study of Double Monsters, and Single Deformities, and the Interaction Among Embryonic Organs During Their Origin and Development, Am. Journ. Anat., viii, No. 2, 1921.

——— Alcohol a Factor in Eliminating Racial Degeneracy, Am. Journ. Med. Sc., clxvii, 469, 1924.

Stoddard, J. L. A Case of Open Ductus Arteriosus with Autopsy, Arch. Int. Med., xii, 38, 1915.

Sutherland, G. A. An Address on Cardiac Problems in Adolescent Life, Brit. Med. Journ., 496, March 24, 1923.

Swanberg and Haynes. A Case of Mongolism in One of Twins, Arch. Neurol. & Pychiat., i, 717, 1919.

Thompson, J. Congenital Mental Defects in Childhood, Diseases of Children, Garrod, Batten and Thursfield, 880, 1913.

Van Slyke, D. D. Gasometric Determination of the Oxygen and Hæmoglobin of Blood, Biol. Chem., xxxiii, 127, 1918.

Vierordt, Hermann. Die Angeborenen Herzkrankheiten, Nothnagel's Spec. Path. u. Ther. xv, Sec. 1, Part 2, 1898.

Warthin, A. S. Congenital Cardiac Syphilis, Am. Journ. Med. Sc., March, 1911.

Warthin, A. S., and Snyder, E. J. Localization of Spirochaeta Pallida in the Heart Muscle in Congenital Syphilis in the Absence of Histologic Lesions or Spirochaetes elsewhere in the body, Journ. Am. Med. Ass., lciii, 689, 1912.

——— The New Pathology of Syphilis, Harveian Oration, Dec. 8, 1917. Published in Harvey Lectures, 1917-1919, with full bibliography of this Author's writings on this subject.

Wells, H. G. Persistent Patency of the Ductus Arteriosus Botalli, Am. Journ. Med. Sc., Sept., 1908.

Williams, F. H. Boston Med. & Surg. Journ., clxxxvii, 412, 1922. Quoted by P. D. White.

Zuntz, N. Physiologie der Blutgase und des Respirationshen Gaswechsels. Hermann's Handb. d. Physiol., 242, 1882.

DISEASES OF THE BLOOD VESSELS

CHAPTER XVI

THE TREATMENT OF ARTERIOSCLEROSIS

Malcolm Goodridge

Definition.—Arteriosclerosis is a pathological term which has been generally accepted to connote changes in the arteries, characterized by hyperplasia, fibrosis, and degeneration of their coats, distinguished clinically by a variety of signs and symptoms due to disturbances in function in organs or other structures supplied by these arteries. Other terms than arteriosclerosis, such as atherosclerosis and endarteritis deformans, have been suggested; but there can be little advantage in substituting such terms for the one which has stood the test of time, and which, moreover, has firmly entrenched itself in the minds of medical men.

Types.—From the standpoint of etiology and symptomatology, I know of no better clinical classification of the various manifestations of this disease than that proposed by Sir Clifford Allbutt. There is a form which seems to be definitely enough associated with various infections and toxic conditions (1) a toxic or infectious type. Another form seems to be related to the declining years of life (2) the senile type—the involutionary or decrescent type of Clifford Allbutt. Still another form is apparently associated with hypertension (3) the hypertensive type. And, finally, one could describe a fourth form, made up of various combinations of the other three, which we might call (4) a mixed type.

Etiology.—We are obliged to concede that comparatively little is known concerning those agents which bear a specific causative relationship to arteriosclerosis, but we must supplement this confession of our ignorance by the statement that a good deal is known concerning those factors which apparently contribute to the etiology of this disease. Old age may not actually produce arteriosclerosis, but old age may well be a predisposing factor, rendering the walls of the arteries more susceptible to pathological change. Hypertension in itself is sufficient to cause changes in the arteries of at least some people. Arteriosclerosis is often enough associated with diabetes, gout, and lead poisoning to arouse the suspicion that there is a cause-and-effect relationship between the general condition on the one hand and the arterial changes on the other.

Certain infections, notably syphilis, rheumatic fever, typhoid fever, influenza, and diphtheria, are capable of producing definite pathological changes in the arteries; and it is quite possible that the tonsils, teeth, sinuses, gall-bladder, appendix, prostate, or pelvic organs may harbor foci of infection responsible in some measure for coincident sclerosis of the arteries.

Marked changes in the arteries of the extremities of laboring men would lead one to ask if there were not some connection between overuse of the member and the accompanying degenerative changes in the vessels. It has been suggested by Sir William Osler that "shoddy" arteries may be a predisposing factor, and that the quality of the tubing may influence both the incidence and the severity of the disease.

High protein feeding in rabbits has produced a lesion not unlike the arteriosclerosis seen in man. Indeed, as far as I know, this is the only instance where a lesion in the arteries has been produced by animal experimentation even simulating the changes seen in the arteries of human beings. Much more vague and less convincing, as causes of arteriosclerosis, are obesity, plethora, endocrine disturbances, and even wear and tear. There is little or no proof that alcohol, tobacco, tea, or coffee is even remotely connected etiologically with disease of the vessels.

Pathology.—This is not the place for a review of the various theories and conceptions concerning the changes which take place in the walls of the arteries in arteriosclerosis, and I shall simply emphasize those features which must be taken into consideration, in order that therapeutic procedures may be instituted on a rational basis. The arteries of the body, large and small, may be pretty generally involved; or the disease may be localized to the vessels of some special organ. For example, it is quite possible to have advanced changes in the arteries of the brain, with little or no change in the peripheral arteries. It is important to remember that we are concerned not only with the changes which take place in the vessel walls, but also with the changes in the organs supplied by the vessels and the disturbance in function of the particular organ contingent upon the interference with its blood supply.

Thickening of the intima is the most common feature of all types of arteriosclerosis. In the senile type, intimal sclerosis is associated with atrophy of the media. Both small and large vessels are involved. The lumen of the smaller vessels may be obliterated, leading to softening of the part supplied by the vessel. In the larger vessels, there is thickening of the intima with deposits of fat, and later necrosis with deposits of cholesterin crystals and calcium salts; these changes may lead to thrombosis, or even to the formation of emboli.

In the infectious type, the media is, or may be, attacked first. There is medial degeneration with intimal proliferation. The streptococcus is quite apt to cause intimal proliferation. In syphilis, infection of the aorta takes place during the late primary stage, causing, after a variable period, granulomatous degeneration of the middle coat, with overgrowth of the intima. In essential hypertension, there is hypertrophy of the muscles of the arterial wall, with proliferation of the intima and degeneration of the deep layers. Diffuse sclerosis tends to affect the smaller arteries. The arterioles are thickened and tortuous. There is fatty degeneration of the intima and thickening of the media, due to hypertrophy, with little tendency to calcification. In Mönckeberg's sclerosis, there is extensive degeneration of the media, followed by calcification and in some cases even ossification. In this type of sclerosis, the changes in the intima are insignificant.

Anatomical changes in the arteries of the brain, kidney, or heart may so reduce the amount of blood reaching the organ that marked changes in structure occur: areas of necrosis in the brain, due to the obliteration or rupture of capillary vessels; glomerular degeneration, fibrosis, and diffuse or patchy atrophy of the cortex of the kidneys; and areas of necrosis, with myocardial degeneration, when the coronary arteries are seriously involved.

Symptoms.—The clinical features of arteriosclerosis depend upon the distribution of the process, with reference to some special organ or tissue; and also upon the extent to which the arteries are involved. The type of the disease will determine, in considerable measure, its clinical manifestations. The symptoms may be general and somewhat vague, or they may attract attention to the brain, heart, or kidney, as the case may be. The onset is apt to be insidious. The patient complains of easy fatiguability, nervousness, irritability, sleeplessness, depression, loss of memory, vague digestive disturbances, cramps in the muscles of the legs or a sense of weight in the lower extremities on walking, dyspnea on exertion, or palpitation. Later, or possibly among the earliest manifestations, there is evidence that the vessels of the brain are particularly involved, and the patient complains of vertigo, headache, continued or paroxysmal; there is loss of mental keenness; slow cerebration; loss of memory, particularly for recent events; emotional instability; nocturnal restlessness. There may be transient attacks of aphasia; convulsive seizures; periods of coma, sometimes mistaken for uremia; as a terminal event in essential hypertension, cerebral apoplexy with hemiplegia. It may be the coronary circulation which is interfered with, and there are attacks of angina pectoris; or a limitation in the ability of the heart to respond to ordinary effort, due to narrowing of the coronary arteries, and, in consequence, diminished blood supply to the heart muscle, with precordial distress, breathlessness, and pain.

With the raised blood-pressure in the hypertensive type of arteriosclerosis, the heart is hypertrophied. Sooner or later, evidences of cardiac defeat creep into the picture. The pressure falls, the amplitude of the pulse is diminished; there is precordial discomfort, dyspnea on exertion, nocturnal dyspnea, dependent edema, passive congestion of the viscera, pulmonary edema, and death from cardiac failure.

The symptoms referable to the abdominal region are often vague: indigestion, nausea and vomiting, visceral crises, and severe pain.

The kidney may show evidences of depressed function; nocturia occurs; polyuria and albuminuria are present; and true uremic manifestations may rarely terminate the disease.

With the involvement of the peripheral vessels, the palpable arteries are thickened and possibly tortuous and beaded; the skin becomes pallid, thin, and attenuated, resembling tissue paper; the feet become cold and painful on walking; there may be a sense of weight or severe pain on exercise; the medium-sized arteries may be gradually obliterated, and gangrene results.

TREATMENT

There is nothing specific which can be done to cure advanced arteriosclerosis, inasmuch as we lack definite knowledge as to the causes of this disease, and because of the very nature of its pathological lesions. Treatment must therefore resolve itself into an effort to delay the progress of an already established disease, and to relieve the symptoms due to disturbance in function in the organs or tissues affected.

I shall deal very briefly with the treatment of syphilitic aortitis and arteriosclerosis of the cerebrospinal system, the coronary arteries with angina pectoris, the renal vessels, and that associated with so-called essential hypertension. For more complete discussion of the treatment of these manifestations of arterial disease, the reader is referred to the appropriate chapters in this section.

General Management

Diet.—In selecting a diet for the patient suffering from arteriosclerosis of any variety, we take into consideration the general condition of the patient, in terms of nutrition; the state of the kidney, expressed in terms of its function; and the condition of the digestive tract, in terms of power to fulfill its function. In anemic and undernourished individuals, a full, nourishing diet should be prescribed; while, in the obese, a reduction cure should be instituted. The minimum diet which will preserve the mental and physical vigor of one suffering at once from obesity and arteriosclerosis is a desideratum. Patients with hypertension, especially, should have the quantity of food limited. It is more important that these patients should not overeat than that they should avoid special kinds of food. There is no reason why an individual without retention of nitrogen, or other evidence of impaired renal function, should be given a diet low in proteids. There is no proof that high proteid intake bears any relation to the incidence of arterial disease in human beings. Limitation of proteids has had no effect in lowering blood-pressure in hypertensive cases; nor has high proteid feeding increased blood-pressure to any marked degree. There is no reason why the patient with no retention of nitrogen may not be allowed a single portion of meat and one egg a day, and it makes very little difference what variety of meat is chosen. Meat-stock soups, because of their high extractive content and poor food value, should be forbidden. The amount of salt taken should be limited to that which is cooked with food. To this end, patients should be instructed not to use salt as a condiment at the table.

In the absence of edema, or other evidence of circulatory failure, there is no reason why the intake of fluids should be greatly restricted. Indeed, there is very good reason why it should not be. Certainly, the patient should have fluid enough to quench his thirst.

A diet low in lime salts has been suggested. Such a diet would mean the restriction of eggs, milk, and vegetables. It is doubtful if deposits of lime salts in the arteries add greatly to the harmful effect of the pathological process; and, at any rate, there are plenty of sources of supply available within the body, even though the diet were quite free of calcium.

Habits.—Tea and coffee should be taken in moderation. There is no good reason why the average patient should not have his cup of coffee in the morning, if he is accustomed to it. If, for any reason, coffee is contraindicated, one may substitute one of the so-called caffein-free preparations.

While there is little evidence to show that alcohol and tobacco cause arteriosclerosis, smoking, in some persons, does cause palpitation, cardiac irregularity, and a rise in blood-pressure; and such individuals should discontinue the use of tobacco entirely, or, at least, greatly restrict its use. In any case, a patient suffering from arteriosclerosis should be cautioned to use tobacco in moderation, and inhalation should be forbidden. Alcoholic drinks should be interdicted, especially in those who have taken alcoholic beverages before meals, to stimulate the appetite. It is conceivable that the extra amount of food eaten under these circumstances is more harmful than the alcohol itself. In aged and debilitated individuals, or in those whose appetites fail, light wines with meals may serve to stimulate a jaded appetite.

Bowels.—The bowels should be regulated by the use of saline cathartics in the morning, or a vegetable cathartic at night. They serve a double purpose: in ridding the intestine of toxic substances and in preventing straining at stool, which, in the presence of hypertension, may so increase pressure in the brain vessels as to produce a cerebral apoplexy.

Exercise.—The amount and kind of exercise to be prescribed will depend on the age of the individual, the stage of the disease, and the efficiency of the circulation. All forms of exercise which put a sudden strain upon the heart and vessels,—sharp, sudden effort, should be avoided, especially in the hypertensive type of the disease, where sudden rise in blood-pressure incident to such effort may produce an apoplexy. The early effect of active exercise is to raise systolic and diastolic pressure. Later, dilatation of the peripheral vessels occurs, and the pressure falls; the relaxation of the skin vessels, following exercise, reduces blood-pressure and relieves the heart. Such exercise as golf, walking, and horseback-riding, is to be recommended, short of undue fatigue or marked dyspnea. Patients should be cautioned against walking in the face of high winds, hurrying, lifting heavy weights, taking exercise directly after meals, and against any form of exertion which causes substernal pain or a sense of constriction in the precordium.

Massage.—In individuals who are unable to take active exercise, massage and passive exercises may be substituted. Such therapeutic measures will be found of more value in the senile type of arteriosclerosis than in the hypertensive type. Massage improves the tone of the skin. Employed on the limbs and splanchnic areas, it improves the movement of blood. It is obvious that such measures are hardly necessary in the type of arteriosclerosis that is associated with hypertension; the walls of the arteries in this type do not need stimulation and exercise.

Hydrotherapy.—Warm baths dilate superficial capillaries, thus promoting the action of the skin and improving elimination. Such baths should be given with great care in hypertensive cases, and particularly in those cases which are associated with symptoms of angina pectoris. The bath should be given as nearly as possible at a neutral temperature, that is, near the temperature of the body, 98° F. to 100° F. Sweat baths, hot-air baths, and steam baths raise blood-pressure, and should be given with extreme caution in cases of arteriosclerosis with hypertension. Mud and peat baths are said to reduce blood-pressure and accelerate the flow of blood through the periphery; and warm saline baths are said to have a more beneficial effect in reducing arterial blood-pressure than the simple baths. Douches, especially cold douches, are to be regarded as cardiac gymnastics, and should be forbidden. Cold baths are contra-indicated in patients suffering from hypertension.

Electricity.—The high frequency current is said to lower peripheral resistance and hence the blood-pressure. One may find excellent authority for its employment in the treatment of hypertension, though, in my experience, I have seen little benefit derived from its use. Humphries suggests that the current be run at 1,000 ma. for fifteen minutes, in daily sittings at first; later, the frequency of the sittings is controlled by the course of the case, indicated in terms of a rise in blood-pressure.

Electric light baths, if given at all, should be given with great caution. All the lights in the cabinet should not be turned on at once; the rapid rise in temperature to 100° F. or more causes contraction of the peripheral vessels and increase in blood-pressure, while, if the lamps are turned on in series and the temperature in the cabinet is gradually raised, dilatation of the peripheral vessels will take place and blood-pressure will fall.

Radium.—That waters containing radium emanations are of any value is at least open to question. It is claimed that such waters lessen the viscosity of the blood, increase diuresis, and increase the elimination of uric acid, and that they lower blood-pressure. The proof that they accomplish the latter rests on very slim evidence.

Occupation.—An individual suffering from arteriosclerosis, who has led and is leading a life of high tension, should be advised to curtail his activities in the field of business; but under no circumstances should a patient suffering from moderately advanced sclerosis without serious complications be advised to discontinue his life work or occupation; such individuals are easily depressed,—lose their interest in life, or, as one man has expressed it, lose their will to live. They should be urged to cultivate a peaceful life, relieved, as much as possible, from strain and worry and excitement. Their work should be restricted, both as to kind and time. They should be encouraged to take long vacations, seeking a warmer *climate* during the cold winter months, the cool climates of the north in the hot summer. Sudden changes in climate, however, are to be avoided, especially sudden change from a warm to a cold climate. Great benefit obtains from the out-of-door life which the warm climate in the winter and the cool climate in the summer encourages.

Rest and Sleep.—Easy fatiguability and sleeplessness are common early manifestations of all types of arteriosclerosis, and are so closely related to one another that avoidance of the one will often prevent the occurrence of the other. Rest is to be encouraged, and by rest is meant mental as well as physical rest.

One cannot make fixed rules as to the amount of time to be spent at rest by a given individual, but fatigue is to be avoided, and it can be anticipated in a measure by advising definite rest periods during the day—an hour after each meal, and at least nine hours in bed a night.

An effort should be made to promote sleep by simple physical measures before resorting to hypnotics; a warm bath at a temperature of 98° to 100° F., just before retiring, followed by a glass of warm milk; gentle rubbing, not stimulating massage, may be helpful in inducing sleep. Where simpler measures fail, we are forced to resort to hypnotics; the smallest doses which will produce the desired effect are to be used, and the preparations selected may with advantage be changed from time to time.

The drugs which have been employed to promote sleep are legion. Among the most valuable are the bromides and certain synthetic compounds containing bromine, paraldehyde, and the members of the barbital group.

Medicinal Treatment

There is no specific treatment. The use of iodids in the treatment of arteriosclerosis has been handed down through successive generations of medical men; and whatever of value there may be in the traditional employment of a remedy in disease, they have, and that is about all that can be said in favor of their administration in arteriosclerosis, excepting cases where syphilis is the etiological agent. It is said that they diminish the viscosity of the blood; but, even when pressed to the point of iodism, they fail to bring about any fall in blood-pressure. They are quite worthless in removing the anatomical effects of arteriosclerosis. Their use, moreover, has the disadvantage of upsetting the digestion. If used at all, they should be used over long periods of time.

Sodium iodid is a little less apt to upset the digestion, and is perhaps a little less likely to cause symptoms of iodism than potassium iodid. The dose of either preparation should be 0.3 to 1.0 gm. (gr. v to gr.xv), three times a day, well diluted with water; or it may be administered in a carbonated water, such as vichy or carbonic.

Other *iodine salts* which are quite as efficacious and less liable to produce gastric disturbances may be utilized as substitutes for sodium or potassium iodid.

Fibrolysin has been suggested, but there is no proof whatsoever that it causes absorption of pathological connective tissue; and, even if it did, I seriously doubt if it would be desirable to use such a preparation in this disease.

Theobromin is said to reduce blood-pressure by splanchnic dilatation.

The use of vasodilators of the *nitrite group* has a real place in the treatment of arteriosclerosis. The widening of the lumen of arteries induced by their administration lowers blood-pressure and mitigates those symptoms which depend upon hypertension. Not only do the nitrites lower blood-pressure, but they also relieve arterial spasm and symptoms incident to such spasm. In susceptible individuals, the nitrites, if given in too large doses, may cause unpleasant symptoms, such as vertigo, nausea, and throbbing headache. It is for this reason advisable to begin the administration of the preparation selected with a small dose.

The preparations commonly used are:

Amyl nitrite, which is usually given by inhalation. It comes in small pearls containing the required dose, 0.2 to 0.3 c.c. ♏ iii to ♏ v). One of these is broken in a handkerchief or towel and held to the patient's face. The effect is rapid, but passes off rapidly.

Nitroglycerin, in doses of 0.0006 gm. (gr. 1/100) in tablets, or 0.03 to 0.2 c.c. (♏ ss to ♏ iii) of 1 per cent alcoholic solution, is rapidly absorbed, but its effects are short-lived.

Sodium nitrite, in doses of 0.06 to 0.12 gm. (gr. i to gr. ii).

Erythrol tetranitrate, in doses of 0.03 to 0.06 gm. (gr. ss to gr. i), given in tablets or capsules, produces the same effect as nitroglycerin and amyl nitrite, but acts much more slowly and for considerably longer periods of time.

The symptoms which occur with episodal crises of hypertension, especially in elderly people, such as headache, restlessness, and sleeplessness, are often controlled by the administration of *chloral,* in doses of 0.3 gm. (gr. v), every four to six hours.

Calomel is said by Clifford Allbutt to be almost a specific drug in hypertension of the essential type. He recommends giving 0.015 gm. (gr. 1/4), one to three times a day, for four or five days, with a saline laxative in addition, testing the blood-pressure frequently during its administration.

In old people suffering from anemia or cachexia, *iron and arsenic* are of undoubted value as tonics.

Symptomatic Treatment

As has already been stated, the symptoms of arteriosclerosis depend upon the disturbances of function in various organs, due to diminished blood supply, contingent upon sclerosis of the vessels supplying the particular organ.

Coronary Sclerosis.—The symptoms of disease of the coronary vessels may be those of myocardial inefficiency, such as precordial discomfort and dyspnea on exercise, or there may be attacks of true angina pectoris; or possibly attacks of cardiac irregularity due to coronary thrombosis may be observed. Briefly, the treatment of angina pectoris resolves itself into the treatment of the attack and the management of the individual between attacks, which is the treatment of the underlying condition, to stay the progress of the arterial disease, if possible.

During an acute attack, and for several days after the acute attack has subsided, the patient should be at rest. The length of time the patient should remain in bed depends upon the severity of the attack; in general, from three to ten days. The dilators of the nitrite group, amyl nitrite inhalations, or nitroglycerin, a 0.0006 gm. (gr. 1/100) tablet dissolved under the tongue, should be administered. Papaverin, an alkaloid obtained from opium, belonging to the benzyl isoquinolin group, 0.03 to 0.08 gm. (gr. ss to gr. iss) is said to have some effect in relaxing spasm in smooth muscle. Mild attacks may be actually aborted by the use of dilator drugs. If the attack is prolonged or severe, a hypodermic of morphin, 0.015 gm. (gr. 1/4), combined with atropin, 0.00045 gm. (gr. 1/150), is to be given to reduce vagus inhibition.

The management of the individual between attacks is that discussed under the heading, General Management of Arteriosclerosis. While complete rest after an attack of angina is necessary, because the nutrition of the heart is disturbed, the rest in bed should not be for too long a period. The amount of exercise permitted must depend upon the response of the given individual's heart to exercise. Exercise must not be of a variety which might initiate a seizure, inasmuch as each attack renders the individual more susceptible to recurrence.

Renal Sclerosis.—The treatment of renal arteriosclerosis hinges upon the function of the kidney. Functional disturbances of the organ depend upon intimate sclerosis of its vessels, or passive congestion due to cardiac inefficiency. The arteriosclerotic kidney associated with essential hypertension rarely gives rise to uremia with retention of nitrogen. In this type of case, the terminal event is usually a cerebral apoplexy or cardiac failure. Should uremia intervene, its management differs in no particular from that discussed in the chapter on Chronic Nephritis. It should be borne in mind, however, that symptoms resembling uremia may occur, due to involvement of cerebral vessels, with the possible addition of some toxic factor as yet but little understood. For symptoms of renal insufficiency, depending on myocardial impairment, such as oliguria, dependent edema, and venous stasis, the treatment is identical with that for cardiac failure under other conditions: digitalis, rest in bed, limitation of fluids, and so forth.

Cerebral Arteriosclerosis.—The symptoms referable to a sclerosis of the brain vessels, such as fatiguability, irritability, slow cerebration, poor memory, loss of mental keenness, with depression and emotional instability, are to be managed with a view to improving the patient's general condition, relieving him, as far as possible, of business worries and anxieties, carefully regulating his exercise, urging long vacations and change of climate, as discussed under General Management.

One observes attacks of paroxysmal headache, with blood-pressures mounting to 250 mm. Hg., in individuals whose average or optimum arterial tension is but 160. Such attacks are often relieved by the use of dilators.

It is possible that some toxic factor is responsible for such episodal crises. We attempt to relieve the effects of toxemia by encouraging the emunctories, promoting diuresis with copious draughts of water, stimulating excretion through the bowels by the administration of saline or hydragogue cathartics; and at the same time we employ dilators of the nitrite group, nitroglycerin, 0.0006 gm. (gr. 1/100) tablet dissolved on the tongue; or erythrol tetranitrate, 0.03 to 0.06 gm. (gr. ss to gr. i) in tablets; or chloral 0.3 to 0.5 gm. (gr. v to gr. x) in solution, repeated in three or four hours.

Sleeplessness and nocturnal restlessness are characteristic symptoms of involvement of the cerebral vessels, especially in the senile type of arteriosclerosis. The milder sedatives, bromids in 1.3 gm. to 2.0 gm. (gr. xx to gr. xxx) doses, or appropriate doses of the soporific group of drugs may be tried. The best of these, in my experience, especially to relieve delirium and restlessness, is paraldehyd, in 15 to 30 c.c. (℥ss to ℥i) doses, in starch water, by rectum; or the 25 per cent elixir of paraldehyd, 15 to 30 c.c. (℥ss to ℥i) by mouth.

Transient vertigo may be relieved by dilators, or by the occasional use of bromids, 1.5 gm. (gr. xx) of sodium or potassium bromid, three times a day, for periods of two or three days at a time.

Patients with hypertension may have transient attacks of aphasia, paralysis, drowsiness, or coma, which last for short periods of time and clear up completely. Such attacks have been explained as being due to claudication of the cerebral vessels, angulation of vessels under high tension, to some toxic factor or factors, or to multiple miliary hemorrhages throughout the cortex. They are usually associated with hypertension, and the symptoms are relieved by such measures as relieve the high tension.

For the management of cerebral apoplexy, hemiplegia, convulsions, the reader is referred to the chapter on Cerebrospinal Arteriosclerosis.

Obliterating Endarteritis.—In obliterating endarteritis, with threatened gangrene, an attempt may be made to establish collateral circulation. Bernheim suggests exercise of the blood-vessels, by alternately plunging the foot into ice water and hot water, leaving the limb in ice water for one or two seconds, then plunging it immediately into very hot water, and repeating this alternation for four or five minutes, ending with heat. This process is repeated three times a day. After each bath, the foot is dried, bathed in oil, and kept warm for an hour. Between the baths, an electric vibrator is used. After two or three weeks, the baths are reduced to two a day. This form of therapy itself gives rise to a certain amount of discomfort, which may be controlled in a measure by the administration of the milder sedatives, such as codein or the bromids. Morphin, because of the duration of the treatment, should not be employed.

In addition to these measures, which have for their object the exercise of the blood-vessels, a method suggested by Moschcowitz, to determine the state of the collateral circulation, is employed in the attempt to dilate diseased vessels. The foot is elevated, and a bandage is applied, beginning at the toes and carried up the leg, with increasing pressure, to a point above the knee. A second, wider, constricting bandage is applied at this level, and the first bandage is released. After a minute, the constricting bandage is suddenly released, and the leg is lowered. This procedure is repeated daily. In patients who are not able to stand such vigorous treatment, the symptoms may be relieved in some measure by rest, electrotherapy, massage, and some form of superheated air. Where gangrene is established, surgical interference must be resorted to.

Intermittent Claudication.—Intermittent claudication, with pain or a sense of weight in the lower extremities during exercise, depends upon interference with the blood supply to the part, due to spasm of the vessels and consequent ischemia. The relief of the pain will be effected by those measures which improve circulation to the part. It has been suggested that an elastic band drawn around the upper third of the thigh, not tight enough to arrest the pulse in the dorsalis pedis, and applied for a period of two hours, twice a day, will cause subsidence of the pain and retrogression of the entire syndrome, if the symptoms are due merely to spasm of the artery.

TREATMENT OF SPECIAL TYPES OF ARTERIOSCLEROSIS

Senile Type.—The treatment of the senile type of arteriosclerosis resolves itself into the management of the patient rather than the condition from which he is suffering. The disease occurs in all conditions of life. Its onset is apparently in no measure determined by manner of living. It occurs in those who lead active lives and in those who take little exercise. It occurs quite as commonly in individuals of temperate habits as in those who indulge their appetites. It is no respecter of sex.

Mental deterioration, loss of memory for recent events, increasing irritability, insomnia, the loss of mental keenness, slow cerebration, marked depression, and, occasionally, convulsive seizures are associated with the senile variety of arteriosclerosis. It is not associated, as a rule, with hypertension, though it may be complicated by an intercurrent hypertension; that is, two types, the essential hypertensive and the senile, occasionally occur together; or it may be punctuated in its course by crises of hypertension. A cerebral apoplexy may take place during a hypertensive crisis. We must deal with these symptoms. With the early manifestations of this type of disease, the patient should be advised to curtail his activities, to take longer vacations and regular exercise. A moderate rise in blood-pressure should not be interfered with. An effort should be made to alleviate symptoms depending upon a diminished blood supply to a particular part, especially the brain. An attempt should be made to improve the general circulation by tonics, iron and arsenic, hydrotherapy, and out-of-door exercise in a climate which is conducive to such exercise.

All possible toxic factors should be eliminated so far as possible by keeping the excretory organs efficiently at work, giving sufficient amounts of water to drink, keeping the bowels freely open.

Inasmuch as patients are easily depressed because of their condition, which they fully appreciate, they should be encouraged by every means at our command, and, while they should be taught to live moderate lives, they should also be assured that the condition from which they are suffering is compatible with a long and useful life. They should avoid mental and physical indolence; but, at the same time, they should be warned against strain of a heart which has undergone degenerative changes.

Toxic or Infectious Type.—In the treatment of the toxic or infectious type of arteriosclerosis, the most important essential is the management of the diseases to which arteriosclerosis may be secondary, and this means the early and effectual treatment of syphilis, lead poisoning, diabetes, gout; the anticipation of possible degenerative changes in acute infections, such as typhoid and diphtheria; careful examination of the arteries in such diseases; the warning of the patient of the necessity for extended rest, or against too early exertion after infection; the control of intestinal toxemia by proper dietary, catharsis, and colonic irrigations; and, finally, careful search for foci of infection, and their removal. Non-syphilitic infectious arteriosclerosis with medial degeneration and definite arterial thickening may disappear, though not necessarily. Hypertension is usually not associated with this type of lesion. In advanced cases of syphilitic sclerosis, little or nothing can be done to cure the lesion, but a good deal can be done, by vigorous antisyphilitic treatment, to relieve symptoms.

Hypertensive Type.—Arteriosclerosis associated with high blood-pressure, if recognized at an early period in the disease, may be cured by a prolonged period of rest in bed. In more advanced stages of the disease, the patient should be taught to live moderately, to avoid excessive strains on his circulatory apparatus, bearing in mind that this type of the disease is prone to run a long course and terminate either in a cerebral apoplexy or cardiac failure. Our efforts should be directed toward delaying these events by properly regulating the patient's life, warning him against sudden effort; specifically directing him to avoid straining at stool, lifting heavy weights, walking against high winds, or going out into the cold improperly clad, taking cold baths, overeating at any meal, exercising directly after meals. All these activities raise the blood-pressure, the increase in intrathoracic pressure is conveyed to the weakened cerebral capillaries, and an apoplexy may result. Patients should not be kept constantly informed of variations in their blood-pressure. Individuals suffering from hypertension can be easily converted into introspective hypochondriacs by failure on the part of the physician to appreciate the necessity for observing this caution.

We must preach moderate living; a diet low in extractives, with moderate protein intake, 75 to 85 gm. a day; reduction cures in the obese; and care of the bowels, kidneys, and skin. We may practice periodic venesection. The dilators may be useful in reducing excessive pressures. The high frequency current may be tried. Failure in cardiac compensation or in kidney function and cerebral accidents will have to be dealt with as they appear. For the details of treatment of these conditions, the reader is referred to the chapter on Essential Hypertension.

CHAPTER XVII

ESSENTIAL HYPERTENSION

Frank Sherman Meara

Introduction.—At the present moment, we know nothing of the cause of essential hypertension or hyperpiesia. The achievement of the last decade in the field of elevated blood-pressures was an appreciation of the fact that the vast majority of cases of increased arterial tension running a chronic course and presenting in their urinary findings a trace of albumin and occasional casts were not, as heretofore they were considered to be, cases of Bright's disease. When the conception of this group as a clinical entity had been accomplished, to which the name "essential" or "primary hypertension" or "hyperpiesia" was attached, many inconsistencies had been cleared up, a new prognosis established in cases of hypertension, and a therapy determined on new principles.

In this chapter only chronic hypertension will be considered and from this group will be excluded that of thyroid origin.

A generation ago we were taught that an increased blood-pressure was indicative of one of two conditions: a chronic nephritis in the clinical sense that the term Bright's disease connotes to both the profession and the laity, or an arteriosclerosis; and the differentiation between the two was determined in the mind of the practitioner by the presence or absence of albuminuria or palpably thickened arteries in those cases not yet advanced to the stage of uremia or arterial accident.

Hypertension under the older view was looked upon as a result, not a cause, of the conditions with which it was associated and slowly but surely it was forced upon observant physicians that in many instances high blood-pressure preceded the appearance of the albuminuria of Bright's disease, and one began to talk of the prealbuminuric stage of nephritis (thus Mahommed). Again it became evident that in many cases of hypertension, which in the absence of albuminuria were ascribed to arteriosclerosis, there was no obvious arteriosclerosis; so one was forced to the assumption of a "latent arteriosclerosis," or a "presclerotic stage of sclerosis," as von Basch termed the one and Huchard the other. Another confusing fact was that the most common and readily determined arteriosclerosis, obtaining in the elderly, was not peculiarly associated with high blood-pressure, and finally that the hypertensive albuminurics did not always go on to uremia and lived an unconscionable long time to the confounding of the traditional prognosis; and, indeed, as this fact was recognized and the protest in the mind of the practitioner against such violations of "the rules" died away, it was recognized that the majority of hypertensive albuminurics failed to "play the game."

At the present day chronic hypertensives are divided into two classes (barring the thyroid cases and a few rare conditions). One group is ascribed to true clinical nephritis or Bright's disease; the other, which constitutes the majority, is due to certain unknown causes, whose earliest operation is manifested as hyperpiesis, to which elevation of pressure other pathological states or functions is in the main attributable. It is to this latter group that this chapter is devoted.

Until we can prove that the *materies morbi* responsible for the hypertension acts detrimentally on other structures, we are warranted in assuming the hyperpiesia, as such, as the determinant of subsequent pathology; but after certain changes have occurred in heart and arteries, the heightened blood-pressure may well enter into a new relationship to these changed structures and the functions they subserve; may well constitute a necessity in some measure; may act in a compensatory capacity.

Results of Continued Hypertension.—Certain results must inevitably accrue to a continued hyperpiesis; first, the heart hypertrophies as one would assume any muscle would do in a response to a continued demand above the ordinary. This is not the place to discuss the quality of this hypertrophied muscle as compared with the normal. It may or may not carry the seeds of weakness; but the fact is that many such hearts remain competent for years, or permanently, while others succumb to increasing demands or too prolonged efforts. Secondly, the arteries undergo sclerosis, due either to the inordinate strain to which they are subjected, or to an unknown toxic agent or both. To the pathological changes in the vessels and the impaired blood supply incident to them are due the disturbances in structure and function of various organs which affords the symptomatology of hyperpiesia. Arteriosclerosis of hyperpietic origin, like the senile type, is a patchy affair and one or more than one territory may be seriously implicated. It is the territorial distribution that determines largely the seriousness of the condition; for though the splenic vessels are commonly affected, the symptomatology is insignificant; but involvement of the coronaries is notoriously rife with danger.

The circulations, which, involved, give character to the picture of hyperpiesia are the *cerebral,* the *coronary,* the *renal* and to a lesser degree the splanchnic and peripheral. From the standpoint of diagnosis and prognosis the rôle of the retinal vessels is unique.

While we do not know the cause of essential hypertension, we are gathering facts about it that may later have an etiological application. In the first place, it is essentially a familial disease. The family history almost invariably brings out a multiplicity of incidence. Parents, grandparents, brothers, sisters, and immediate collaterals will offer cases of apoplexy, angina pectoris, heart failure, kidney disease or other recognized sequelæ to a chronic hypertension. One is impressed with the feeling that the germ plasm of family groups contained a something or lacked a something that conduced to or made a susceptibility to this condition on the part of individuals evolved from it. As one notes the incidence of this disease in the fourth and fifth decades, there is demonstrated in one's mind the conception to which is attached that much-abused term an "abiotic" state.

Again one is impressed with its occurrence in individuals of a certain habitus—the ruddy-faced, short-necked, square-shouldered, thick-chested, short-abdomened, stocky or thick-set type. It occurs especially in men and women of active, energetic, dominating, and executive mental habits. For this reason it occurs more particularly among the higher classes and, though by no means rare in the laboring classes, is distinctly less common. If there is association with an anatomical type, one thinks of the rôle of the endocrines; but it must be emphatically stated that there is no proof at present that endocrines do play a rôle. Unfortunately the undoubted importance of the glands of internal secretion, has led to countless theories of their rôle in disease, and, worse yet, to pernicious and promiscuous usage of gland products in the hands of charlatans and the well-intentioned, but mentally inert, physician.

Like diabetes, hyperpiesia is especially vicious in early life and relatively benign in the elderly.

Then, too, there are certain things that primary hypertension is not. It is not the product of an infectious process; nor is syphilis the direct cause of high blood-pressure and that, too, in spite of the fact that its attack upon the artery is so common and so severe. Hypertensives may well have syphilis and hypertensives may harbor latent foci of infection and suffer material damage from these infections in the very structures that are imperiled by the hyperpiesis, which makes their appreciation and removal a matter of moment; but such removal does not affect the blood-pressure readings.

Again hyperpiesia is not a dietetic disease, at least not so proved; but dietetic factors play no mean rôle in modifying degrees of hypertension in some individual cases. Just what percentage of the adult population has an essential hypertension is not known accurately; but it is a very considerable one and, while the condition is responsible for their death probably in the large majority of cases, the number who enjoy a reasonably long life in years and a longer life in actual accomplishment (for as has been said such people often live intensively) is legion, and they are, may one not say, happily unaware of their condition in the meantime. Many live to threescore years and ten and longer and many die of diseases entirely irrelevant to their circulatory pathology. Some of the greatest men in the world have accomplished mighty deeds while this state obtained.

It is obvious, then, that compensatory efforts on the part of the body result in a readjustment of vital mechanisms, that is, in countless cases, adequate to the demand. This is an important bit of information for the physician to entertain; for by it his attitude toward hyperpietics must in a measure be determined.

It should by no means lead him to a policy of *laissez aller,* but there is just one thing worse than inactivity and that is pernicious interference. By this I mean that it were infinitely better that most of these patients never saw a physician than that every such patient should be considered as threatened with imminent disaster; invalided by his physician; his business disrupted; his family interests imperiled; his mental peace destroyed; to order his goings and his comings, his risings and his retirings, his table and his pleasures by the vacillations of a sphygmomanometer; to ride with Black Care behind his saddle and to sit under the sword of Damocles. This is exactly what happens repeatedly when such cases fall into the hands of physicians ill-informed, unexperienced, and who leave common sense out of their prescriptions.

From the standpoint of treatment one may divide these cases into those who show symptoms and those who do not. The latter group are accidentally discovered to be hyperpietics in the course of an examination carried out for an irrelevant condition; in the course of life insurance examinations, or routine examinations by institutes or physicians of those whose foresight dictates such a procedure. Unless the individual has submitted himself to reëxaminations at fairly frequent intervals it is impossible to tell how long he has suffered with the hyperpiesis.

For those in whom the increased blood-pressure can be determined as of recent origin, Sir Clifford Allbutt pronounces an optimistic prognosis, if they are promptly subjected to treatment; indeed, he says: "If we can catch hyperpiesia early, and keep at work against it, it can be cured more often than not," and further adds: *"When the system has taken a new set, the whole has readjusted to the altered conditions, and the new attitude is more or less permanent. Therefore, to bring back the old equilibrium of pressures by active treatment is out of the question and diligently to attempt it is to do more harm than good."* The italics are mine, but an observation of well over a half thousand cases in my own practice since we have come to recognize hyperpiesia as an entity has confirmed for me the accuracy and wisdom of this quotation. I am, however, unable to confirm the first statement. I am unable to assert positively a cure in any case; but I have seen gratifying amelioration in one class of cases, namely, those who are frankly overweight.

Treatment of Early Cases.—I do believe it wise in every early case, in every case in the first three decades, in any case at any age in which the effort will not be a sacrifice to the patient's interests or implant the habit of introspection and mental concern, so easily established in certain individuals, to test the result of treatment.

Rest.—This will consist of rest in bed, until the optimum reduction in pressure readings occurs, or the confinement defeats its purpose in inducing restlessness, anxiety or distrust.

Diet.—Regulation according to instructions later laid down, the essential features of which are an economical intake and reduction of weight in the obese.

Sedatives.—Mental therapy, elimination of worry, perhaps change of environment, and such drugs as will reduce nervous excitation if such exist.

When desired results are obtained or the maximum that can be expected under the circumstances, a gradual return to normal or restricted activities under observation and then a regulation of life according to results is indicated.

During such treatment blood-pressure will almost invariably fall and cardiac overactivity decrease; but my own experience has been that the "new set," as Allbutt calls it, has already occurred and the pressure readings rise as activity increases, though not infrequently a distinct betterment is noted. However, Allbutt's testimony alone almost compels the effort and his results must be accepted as a basis of hope for ultimate success.

Treatment of Later Cases.—A word about blood-pressure readings. Certain precautions are to be taken. Blood-pressure is a function in circulatory efficiency; hence, every varying demand on the circulation is accompanied by variations in the blood pressure. Recent physical exercise, recent mental excitation, a recent hearty meal make the blood pressure readings unreliable.

A patient coming to the office at the end of a brisk walk will not give the same reading as after a rest. Men after an exciting day in their business or on the floor of a Stock Exchange, seeking an examination for life insurance are often held up for an abnormally high blood-pressure, which after a night's rest is found normal. For this reason the first examination in a doctor's office may be accompanied by a perturbation that renders the reading unreliable. Observations on the same individual should be made under much the same conditions in order to interpret results in terms of measures instituted. The patient should be relaxed and pay no attention to the proceedings. Watching the dial or column of the sphygmomanometer is accompanied by the usual muscular contraction of attention and elevates the reading a few points.

Some arteries contract locally, that is, go into spasm under the pressure of the cuff when the first reading is made and more especially if insulted by a sudden and unnecessarily high inflation. The inflation should be made gradually and the readings repeated at each sitting until they are constant. Often the artery seems to undergo a slow cycle of contraction and dilatation that makes a definite systolic and diastolic reading impossible. If one holds the pressure at the first reading of either systolic or diastolic pressure he will hear a waxing and waning of the sounds through a full 10 mm. All these facts show how absurd it is to lay stress on such variations from day to day; and yet the sun is made to shine or the clouds to collect for that day for many a patient by a difference of 10 mm. in the reading.

It should be remembered that variation in blood-pressure in response to a given stimulus is far greater than in the normal individuals; in other words, the blood-pressure mechanism is unstable, much as the heat regulating mechanism is unstable in the presence of certain toxins of infecting organisms.

Calcified arteries are a snare for the unwary, for no accurate reading can be made below a badly compressible artery. I recall a man who went from physician to physician as a curiosity, having a blood-pressure beyond the powers of registration of any apparatus at hand. He had brachials that were as incompressible as brass tubes.

A more common pitfall is the very small artery of a stenotic mitral-valve or the less common aortic. In these cases the finger judges a low blood-pressure so low that the physician thinks it idle to take the reading and if he does may fall a victim to the silence of the second phase.

I have never seen more astonished countenances than those of physicians who read a systolic pressure in a mitral stenosis as 120 when shown that it was well over 200 and that the first phase had been missed because no such pressure had been applied to the cuff and this phase was followed by a second in which silence obtained until the thump of the third phase was heard and interpreted as the first.

It is well known that no inconsiderable number of mitral stenoses, though a small minority of the whole, are accompanied by a high blood-pressure. Whether this is due to renal changes induced by passive congestion or is a coincidence of the two conditions is not yet definitely settled.

Finally one should beware of eliminating hyperpiesia in a case of failing myocardium, if there is definite cardiac hypertrophy and no valvular disease; for the failing myocardium may be responsible for the low reading.

It seems no longer necessary to emphasize to the profession at large the necessity of reading both systolic and diastolic pressures; but it is not supererogatory to insist that of the two readings the diastolic is the more important. The prognosis varies largely with the diastolic pressure.

Patients with a low diastolic pressure are dealing with a relatively benign condition compared with those in whom the diastolic is consistently high. We may then divide roughly hyperpietics into two classes: those with a low diastolic and those with a high diastolic.

Hyperpietics with Low Diastolic Pressure.—In the adult the average systolic pressure is 120 mm. Hg., and it does not vary much with advancing years. Variations of some 20 mm. above and below this figure may still be considered normal, though a steady approximation to either extreme must be looked upon with suspicion. I take 160 and above constantly maintained, as hypertensive, 140 to 160 as probably such. From the diastolic average of 80 mm. lesser variations are expected; a diastolic of 90 mm. or above is hypertensive.

The systolic pressure, of course, is the pressure under which the artery finds itself at the height of the impulse of blood driven into it by the cardiac systoles; it is then a function of cardiac output. If the diastolic remains normal, the artery stretched by an increased content during systole finds repose during the succeeding diastole. Its period of rest is unimpaired. In these cases it would seem as if the excitant operated on the musculature of the heart, but spared that of the artery. When the diastolic pressure is increased, one must assume an excitation of the muscle coat of the arteries or of the contractile tissues of the arterioles. The strain is never quite relieved and the artery fails to get its desired amount of rest. Hypertrophy of muscle coats ensues and later the degenerative processes that constitute arteriosclerosis. However inadequate such an explanation may be, the fact remains that high systolic pressures are well endured and may be symptomless for years. Of course, this high systolic pressure and low diastolic pressure give a large pulse pressure (P. P., the difference between the two), and if one takes as a very rough measure of circulatory work the P. P. times R (pulse rate), we see that this is much increased. For example a patient with a normal pulse rate of 70 has a formula of P. P. equals 40, times R. equals 70, equals 2,800. A patient with a reading of $\frac{200}{90}$ and pulse of 70 has a formula of 110 times 70 equals 7,700. This means a tremendous increase in work and is necessarily accompanied by cardiac hypertrophy; but as long as the heart remains good it also means abundant supply to the tissues at large. But I have also been impressed by the fact that when the diastolic is raised, if the P. P. remains large, the patients do relatively well compared with those in whom a high diastolic and low systolic gives a small

P. P. I recall one patient who represented the extreme of this condition $\frac{200}{180}$. The pulse was barely perceptible and the distress extreme.

Patients with High Diastolic Pressure.—These constitute the serious cases we are called upon to treat and those in whom apoplexies, coronary scleroses, renal complications and other sequelæ usually occur. This division has greater prognostic significance than therapeutic; the same measures are applicable to both.

Rest.—Rest is the key-note of the treatment of the hypertension, but rest is a relative term. Rest does not mean, necessarily, abeyance of function; it means economy of function as well. The circulatory system is overworked at both ends. The heart is overworked and the arterial tree is overworked. The heart has hypertrophied to meet the increased demand and so have the muscle fibers and elastic fibers of the arteries. Mental activity, physical activity, chemical activity, increase the circulatory demands. These activities are the expressions of life; with their cessation life ceases, but they can be kept within bounds.

Treatment of Cases without Symptoms.—What shall we say to the man in whom hypertension has been discovered, but of which no faltering of function had made him aware? The procedure advisable in the suspected early case has been stated above; but with these exceptions or when thought feasible one should endeavor to explain the state of affairs with proper optimism, avoiding treating it as trivial. The patient will be immensely helped by the assurance that there is no call to interrupt his activities and will be all the more amenable to suggestions that he should not increase them; that he avoid additional obligations that are not necessities.

One should explain the unhappy effects of continuous mental strain and the value of interruptions by holidays and diversions. One should treat the dietetics of these cases on the broad basis of cutting out excess rather than fixing a rigid diet and one should insist on reëxaminations at intervals of at least six months, provided no symptoms appear in the meantime.

Treatment of Cases Showing Symptoms.—No matter how trivial the symptom which can be directly referred to the hyperpiesia, it means that the patient has entered on a new era; cardiac compensation is threatened, vascular accident is forecast or some organ is receiving an inadequate blood supply. Such is the meaning of unaccustomed shortness of breath after effort, an awareness of the heart action, cardiac oppression or pain, undue fatigue without adequate cause, irritability, increasing difficulty in remembering, mental lag, change of temperament, insomnia, headaches, dizziness, nocturia, gastric disturbances, nose-bleeds, metrorrhagias, hematurias, retinal hemorrhages. Now a more intimate relation is established between physician and patient. The whole case is to be reviewed; advice should no longer be in terms of generalizations, but definite instructions should be given and understood. The physician must become the doctor in the original sense of the term, the teacher. He will find that time taken for a simple explanation of why he asks the patient to do or not to do this or that will reconcile the patient to many measures the wisdom of which he would otherwise question.

Habits of Life.—In the first place we should spend sufficient time to understand the nature of our patient's activities; how long he has exercised them; how he reacts to his obligations; his temperament. Because a man is at the head of a vast industry or because he is identified with a multiplicity of important interests does not necessarily mean that he is grossly overworked. He may have his work so organized or so well in hand that he accomplishes maximum results with minimum efforts. To another man a seemingly trivial demand may carry with it anxiety, depression and a threatened breakdown. Other things being equal, one may say that these patients do better if they retain their interests and do some work than if they give them up. This does not mean that a period of complete rest for a time to restore compensation is not desirable, for it most assuredly is; but it means that the relinquishment of work to which habit has accustomed them is not infrequently followed by rapid deterioration. They fail to adjust themselves to new conditions. An exception to the rule holds in the case of those individuals who have happily established outside interests and avocations in which they may take refuge.

With such a period of rest or without it one proceeds to lay down rules of conduct. Let us take for example a business man.

He should be advised that he may continue his work. Such reassurance has a most happy psychic effect. He is asked, however, to take on no new enterprise to which he is unaccustomed and which means intensive work to accomplish, worry about its ultimate success and the finances involved. He should be advised to shorten his hours at the office; to go down at a later hour and return earlier. He should be advised to cultivate the habit of week-ends and utilize them for the purposes of rest and recreation. He should be advised to interrupt his work more frequently for short vacations and trips and to have a longer and real vacation at least once a year. He should be advised to lie down in the middle of the day for a few minutes to sleep or at least relax. He should be urged to spend more hours in bed each night and to rest a while after the evening meal, so often the heartiest of the day.

Clothing.—One should clothe to keep warm, but not overload; light woolen or silk next to the skin takes up the moisture and preserves a more even temperature than cotton or linen. He should not expose himself to the sharp cold of winter even for brief periods without adequate overclothing. He should endeavor to preserve even temperatures in his office and in his home; avoid overheated and crowded conveyances which increase the hazard of infection. He should sleep in a room with fresh air; but avoid exposure during the night, and more especially if a restless sleeper by wearing pajamas or night dress of woolen or containing a goodly percentage of wool; this does not mean heavy garments.

Bathing.—If he has had the lifetime habit of cold baths and reacts well to them they may be continued; theoretically very cold and very hot baths are less desirable to hyperpietics than warm baths; but if warm baths be indulged in, they were better taken at night or sufficiently long before going into the cold for the superficial vessels to regain their tone.

Exercise.—Again previous habits must be considered, the state of the heart, and the studied reaction to exercise. Some form of exercise is desirable; for the more sedentary, walking or not too strenuous golf, or horseback-riding; to those who have indulged in more active forms of exercise and sport, the same may be continued after suitable observation of their reaction toward them. Excessive and sudden effort should be avoided, as cardiac strain or a ruptured cerebral vessel may result. Excessive mental excitement should be avoided: anger, altercation, public speaking for those unaccustomed, or positions of mental embarrassment.

Diet.—Very different ideas have been entertained in regard to dietetic restrictions in this condition. The truth of the matter is, a diet may not yet be laid down on the basis of sound and accepted principles. Few, if any, of us believe that diet is the primary cause of hypertension; but most of us believe that, in some cases at least, diet does modify the hyperpiesis.

Quantity.—Quantity seems to me to play the most considerable rôle. Patients with essential hypertension have, as a rule, an unusually good appetite which they indulge without stint. They are intemperate of food rather than drink, and alcoholism, in my experience, has not been common among them. Alcohol has been used rather as a contributor to the feast than to the debauch. Of course, with a break in the circulatory mechanism appetite falls away. These patients eat too much. They consistently exceed their protein and caloric requirements.

Now the gospel of therapy in hyperpiesia is rest; rest of the numerous functions concerned in digestion, circulation, metabolism and excretion is indicated. Every increase of function, wherever and however exercised, means more work for the overstrained circulatory apparatus and, even if the elements of food in excess were innocent of harm, the economy of effort attained by diminution of diet is a desideratum.

If a patient is overweight, an effort should be made by restricting food intake to approximate a normal weight for a given stature and age. An excess of adipose is an additional burden to carry about, requires more work and more circulatory activity and, while the blood supply to fat is relatively small, it nevertheless must be maintained.

Two things must be kept in mind in reducing weight: First, it must be done gradually. Violent reductions by very scant diets are not infrequently followed by depression, weakness and obviously disturbed functions. Second, it must be remembered that the storage of fat is not entirely determined by food; that the endocrines play a rôle and that the laying on of fat in some cases seems entirely disproportionate to the amount of food ingested and that reduction of diet may be followed by disappointing persistencies in fat retention and a weakening of the patient that makes us feel that the fats and carbohydrates have been diverted from their active physiological functions to the inactive fat depots. Certain it is that many patients look and feel best when frankly defying the standards of weight. Common sense is as important an ingredient of their dietaries as calories and vitamins.

Proteins.—It has long been assumed that high proteins, and especially animal proteins, played a deleterious rôle in hyperpiesia; that the pressure was aggravated or maintained by the ingestion of meats and that perhaps there were other obscure damages wrought by them.

Chittenden showed some years ago that the accepted standards of protein needs determined by studying the customary intake of different races, individuals, and occupations were higher than could be shown necessary to maintain a considerable number of carefully observed active young men in vigor and activity. Even more extensive and detailed experiments, including calorimetric studies were carried out by Benedict on a group of Y.M.C.A. students at Springfield, Massachusetts. These men, leading an active, even athletic life, were maintained on 10.5 gm. of nitrogen (68.5 gm. protein) per day against 14 gm. ingested by a control group with unrestricted diet. At the same time they were maintained on 1,950 calories, little more than one-half the amount originally required. Pulses were slowed to 30 to 33 when taken in the morning and both systolic and diastolic pressures were distinctly lowered. Benedict was showing that in time of war dietetic restrictions were justified and even commendable. He held no brief against the usual ingestion under ordinary circumstances; nor did he draw conclusions as to diet in hyperpiesia.

For a long time I rendered my patients vegetarians; but in the end could not demonstrate that any gain had been attained; but often much unhappiness, distaste for food and gastro-intestinal disturbances.

I do believe, however, that rest of function should be exercised and that protein should be materially diminished. I allow my patients one modest helping of meat, fowl, or fish once a day. If more definite rules be desired, Foster has recommended 0.8 gm. of protein per kg. of body weight, allowance being made in the obviously obese.

Red meat is often condemned as especially bad for hyperpietics. There is little reason for this, except that red meats, such as beef, are savory and excite appetite, and the other fact that is offered for what it is worth—that not a few patients complain of flushing of the head and other discomforts when they indulge in it.

It is desirable, when possible, to have the blood urea nitrogen or nonprotein nitrogen determined; for in those cases in which the kidney has undergone decompensation from intrinsic changes, or in states of pronounced passive congestion, urea nitrogen above the normal limits may be demonstrated. Under these circumstances, a marked reduction of nitrogen intake is indicated until such reduction in the blood occurs as is possible in a given case.

Sugar.—Sugar in the blood of hyperpietics is often found to maintain, even though within normal limits, a high level; this may be true even in those who are underweight. In such cases, that is, when, during a fasting period, blood sugar ranges at 125 mg. per 100 c.c. or above, and all the more when the patient is overweight, I think it wise to reduce the carbohydrates in the diet gradually until such increase in the blood sugar disappears or is evidently normal to the individual. One must remember that in certain rare cases diabetes occurring in a hyperpietic may be assumed to be due to vascular changes in the pancreas, and involvement of the pancreatic arteries is exceedingly common.

Salt.—Recently the effects of salt on hyperpiesia has been much discussed. Years ago Dr. Robert Cooke in a series of unpublished studies in my wards at Bellevue Hospital showed that the ingestion of a sudden but not excessive salt increase was followed in certain cases by a notable increase of blood-pressure in hypertensives; but of late the work of Allen had led him to the opinion that salt played no mean rôle in at least maintaining pressure to some degree beyond that exhibited on a salt-free diet. Efforts to corroborate this experience have led to conflicting results. Among these might be cited some of the experiments of O'Hare and of McLester.

It has so happened that some of my patients have been subjected to a salt-free diet in an institution under the direct observation of an ardent advocate of the measure. Extraordinary falls of pressure approximating normal readings occurred over a period of weeks; but within forty-eight hours after return home and under the same diet carried out conscientiously, I have found the pressure again well above 200 and remaining so; even though the patient persisted in continuing the diet, contrary to my advice. It has been my custom for years, both on the basis of the work done in my wards and because it is well known that the habit of using salt as a condiment leads to an ingestion of salt far beyond physiological needs, and because no stimulant to an already overeager appetite is called for, to diminish the salt intake of my patients. Of late I have been guided by estimations of blood salt. When I have found an excess above 560 mg. per 100 c.c. of blood-plasma I have been insistent on reduction; and I have found these excesses even in patients who show no nitrogen retention. Under all circumstances I advise eliminating the use of salt at the table; that is, as a condiment; but allowing enough salt in the cooking to make the food palatable. Butter should be unsalted; no salt meat or fish are allowed, and, of course, the obvious ham and bacon, which strangely enough is not always obvious to the patient. I also exclude for their salt content, meat soups or soups containing meat stock or meat extracts.

Common sense, as I have intimated, must be the chief ingredient of all dietaries. If food becomes unpalatable, appetite languishes, and digestive processes are disturbed, away with theory! Harm done the patient by persistence is patent; good done is theoretical.

Fats.—In so far as they conduce to overweight fats should be restricted; there is no other contra-indication to their use.

Fluids.—There are observations on the ingestion of large quantities of fluids that tend to show that an increase of blood-pressure follows; but it is hard to prove that an ordinary intake has any such results. My own attitude toward the subject has been to limit the intake to 1½ quarts to 2 quarts a day, but after all to be guided by thirst. If excesses of neither sugar nor salt have been indulged in these amounts are rarely exceeded, if equaled; but it does prevent the drinking of huge quantities of fluids to "wash out" poisons. What is taken in must be put out and that means extra work for circulatory and eliminating organs. In other words, *rest* is indicated in the sense of relief from overwork.

Summary.—For the overweighted, diet is reduced by cutting down carbohydrates and fats to cause a gradual reduction to something like a normal standard; the steady loss of ½ to 1 pound a week will answer. The amount can be regulated by the scales or, better yet, and particularly because adiposity is so persistently retained by some individuals that weakness rather than loss of weight follows too great restrictions, by determination of caloric intake. For the hyperpietic in general, enough, only, and not too much; meat, fish, or fowl, a modest helping once a day; reduction of salt and avoidance of great quantities of fluid.

For cases of suspected renal deficiency, there should be a determination of nitrogen, sugar and salt in the blood as a guide. Avoid exercise immediately after meals. Rest a short time after meals. Take the heartiest meal when the day's work is done but rest before the meal if fatigued. A lifetime habit respecting the time of the heartiest meal must be considered. Digestive disturbances need prompt medical attention; they are most likely to be due to circulatory difficulties. Cases of cardiac decompensation, cerebral accidents, frank renal decompensation or other serious sequelæ of hyperpiesia require special dietetic consideration. Diet is as truly a product of evolution as the organism it subserves; he who modifies it should recall this fact.

CARDIAC INVOLVEMENT

Most of the victims of essential hypertension die of cardiac failure. One gathers the impression that the causes of hypertension are operative on the circulatory apparatus as a whole; that in a certain number of the cases the heart bears the brunt of the attack in the beginning; this is the case with those patients whose diastolic pressure is consistently low, often normal. The heart seems to be driven, to pound, to hurl unaccustomed quantities of blood into the arteries with each beat to raise the systolic pressure. These hearts are in many ways analogous to the hearts of hyperthyroidism. They may be uncomfortable hearts in the sense of the awareness they breed in the patient; but in my experience they are the cases of favorable prognosis as to length of life. They are willing horses, yet suffering the spur. When they cause discomfort, rest of mind by the use of gentle sedatives, such as bromids, for example, mixed (triple) bromids, gr. xv (1 gm.) three or four times a day, or luminal in ½-gr. doses three times a day and mild hypnotics to ensure sleep, will work wonders. As hypnotics, trional, gr. v (0.33 gm.), to be repeated once or twice is my favorite, but adalin, gr. v (0.33 gm.), or chloralamid, gr. xv (1 gm.), or even medinal, gr. v (0.33 gm.), or veronal, gr. v (0.33 gm.), may be used. The last is potent but the least used by me.

In most instances, however, in addition to such effects as the unknown causes have directly on the heart, that organ has to overcome resistance ahead in the shape of contracted arteries, which afford a high diastolic pressure, and the higher it is the worse the prospects for the heart. The heart hypertrophies to meet the increased demand and it meets it with amazing efficiency over often astonishing periods; but at last it falters and then fails.

Early Cases of Cardiac Inefficiency.—Early signs of heart distress from any cause whatsoever may be expressed by shortness of breath on effort, by cardiac pain, by edema of the extremities or by unwonted exhaustion. In this class of hearts, edema, while it may occur, is not the rule; indeed, the heart more often marches up to its last barrier without ever developing edema. The first cardiac manifestation may be a serious one, an attack of pulmonary edema; or the patient arouses from sleep with a sense of suffocation that makes him sit up in bed to breathe, a condition sometimes termed cardiac asthma; or the patient may have an anginal attack.

A physical examination may first detect cardiac incompetency or corroborate symptoms, suspicious, rather than declared; for example, a heart rate in recumbency distinctly above normal, a change in the quality of a first sound that one is familiar with, and more particularly when beside a muffling or a shortening and sharpening of the first sound that gives a tic-tac quality to it, an embryocardia, one gets a splitting or reduplication of the first sound; the rhythm is that of the anapest in versification, two short and a long ⏑ ⏑ — in contradistinction to the dactyl, one long and two short — ⏑ ⏑ of the reduplicated second. It is called when pronounced, a *bruit de galop* and it does imitate to a nicety the beat of the hoofs of a galloping horse. There may also be detected development of murmurs not heard before, especially a diastolic, or of certain arhythmias, such as atrioventricular block, auricular flutter or auricular fibrillation—the last two relatively rare in hyperpiesias—a definite engorgement of the liver and a few râles persistent at the bases of the lungs.

A fall of a long and consistently sustained blood-pressure, accompanied by a lessening of well-being, loss of flesh, fatigue or other symptoms referable to the heart should be interpreted as indicative of myocardial weakening, not as a cause for congratulation, as so often happens; however, it is a peculiarity of these hearts that they frequently maintain excessive pressures to the end, like the "one horse chaise" they give no hint before rapid dissolution. A hypertensive crisis, with striking increase of blood-pressure, may be the first call for interference.

An electrocardiogram sometimes discloses greater damage than might be suspected from the heart's operation: lengthening of conduction time, deformities of the Q.R.S. complexes interpreting degenerative processes in the Purkinje fibers, or changes in or inversions of the T-wave warn of myocardial weakness or fatigue.

Any of these observations should indicate greater restrictions than heretofore observed: An interruption of routine activities for a time; rest, complete or partial, depending on the seriousness of the symptoms and the patient's temperament. Insistence on such dietetic restrictions as are detailed above, or for a time a diet even more restricted; attention to the bowels; mild sedatives in the excitable, and assurance of sleep by mild hypnotics; in the plethoric with hypertensive crises venesection of 12 to 16 ounces, and in almost every case some digitalis support; moderate doses of 6 gr. (0.4 gm.) a day for four or five days and then 1½ to 3 gr. (0.1 to 0.2 gm.) a day as long as needed. When improvement has been attained, a holiday in some new and restful environment before return to work and after that as frequent observations as will serve the interests of the patient without impressing him with a sense of being dangerously ill.

Let us consider some of the early serious cardiac symptoms before we take up a badly broken compensation.

Dyspneas of Acute Onset—*Cardiac Asthma.*—I am not sure that this is a desirable term for the condition about to be described, but it is retained to differentiate it from the forms that follow. It is a sudden shortness of breath, apt to come on not long after the patient has fallen asleep, or in the early morning; it is a sign of cardiac distress curiously associated with rest. The patient awakens and is impelled to sit up in bed to get his breath; to the onlooker the attack suggests a mild or severe bronchial asthma, but on examination does not disclose the tight contraction of the bronchi with its characteristic signs. It is not accompanied by anginal pain. After a time the attack subsides, and it may not be long, and the patient again falls asleep. One sees such attacks in other myocardial disease than that of hyperpiesia, and it has always seemed to me an ominous symptom. It may be taken as an expression of a bad myocardium; it is associated in my mind in its significance with an alternating pulse, though not necessarily so associated in the patient. It seems more a forewarning of impending disaster than of present danger, though I have seen sudden death follow immediately upon it. Such attacks may be provoked by effort. For the immediate attack I know of nothing better than morphin, gr. 1/8 (0.008 gm.) to gr. 1/4 (0.016 gm.) hypodermically.

If possible an electrocardiogram should be obtained as soon after the attack as feasible for such evidences of myocardial involvement as it may afford. Rest in bed on a restricted diet should be insisted on together with suitable digitalis medication.

Dyspnea of Acidosis.—It has been shown that a form of dyspnea simulating cardiac asthma may be due to a relative acidosis, that is, decrease of alkaline bases in the blood. When laboratory facilities will permit of the determination of acidosis, for example, by the Van Slyke method of estimating the CO_2 combining power of the plasma, it should be done; if this is not possible, bicarbonate of soda may be administered in 5-gm. doses, that is, roughly, level teaspoonful doses at two-hour intervals. Two such doses should alkalinize a urine of normal acidity; if more be needed the degree of acidosis may be roughly estimated by the quantity administered to accomplish the result; at the same time an appropriate therapy has been established by the procedure.

Uremic Dyspnea.—The possibility of such a cause for dyspnea should be kept in mind; in the absence of corroborative uremic symptoms one should have recourse to determination of nitrogen retention in the blood and other renal functional tests.

Paroxysmal Dyspnea.—This term may well be retained for those violent attacks of dyspnea with lividity, even to witness which is agonizing. I recall two such cases in my practice. These attacks were paralleled in their intensity by nothing I have witnessed so much as by an anaphylactic reaction of a patient sensitized to horse serum after receiving an injection of diphtheria antitoxin (horse serum); but the distress and terror as the patient threw himself about and struggled for breath was greater in the former than in the latter. In both serum asthma and the paroxysmal dyspnea, the cause is a violent contraction of the bronchioles and the latter is attributed by Longcope, as the result of his studies, to an involvement of the nerve plexuses at the root of the aorta in an aortitis—the same nerve plexuses, I take it, that Allbutt looks upon as the seat of origin of anginal pains.

Nitroglycerin is indicated; the dose sufficient, gr. 1/50 to 1/10 (0.0012 to 0.006 gm.), under the tongue if the patient can retain it and the tongue is moist enough to assume its absorption, which in such cases is unlikely; otherwise under the skin. It is my experience that nitroglycerin is more rapidly absorbed and more effectual when absorbed from under the tongue than when given under the skin and least effectual when swallowed. The spirits of glonoin (spiritus glycerylis nitratis) ♏ i equals gr. 1/100 (0.0006 gm.), is the best form; but it is rarely at hand for the emergency; hypodermic tablets are to be preferred to the tablet triturates unless the latter happen to be very freshly made. Doses should be repeated with great frequency and in increasing amounts. I do not know of any toxic dose or toxic effect outside of the splitting headache that follows an unnecessarily large dose.

In the meantime morphin will be administered hypodermically, gr. ⅙ to ⅓ (0.01 to 0.02 gm.), and repeated if necessary.

Such attacks connote a dangerous state of affairs; but one of my patients had had the attacks for six months and latterly many in a day before he succumbed.

Pulmonary Edema.—This is not an uncommon occurrence in the course of hyperpiesia. It is prone to come on with startling rapidity and often, perhaps usually, without obvious provoking cause. An altogether satisfactory explanation of its mechanism has not been offered, but the most appealing perhaps is an accumulation of blood in the pulmonary circuit, due to disproportion in efficiency in the two sides of the heart; an efficient right heart forcing blood into the pulmonary arteries, while an inefficient left heart fails to empty the pulmonary veins. The first attack may be the last as was the case in a patient found dead in her bed, in so peaceful an attitude that death must have occurred in quiet sleep, the mode of death being attested by the masses of dried foam about the mouth and nostrils. On the other hand, it is a source of surprise how many attacks, each alarming in appearance, a patient may survive.

In the sudden attacks, the râles may not necessarily first appear at the bases as in the cases in gradually failing hearts, but anywhere in the lung; the coarser râles are audible to a bystander. Many attacks clear up without assistance as mysteriously as they appear. Nitroglycerin is theoretically indicated in such an attack, as it dilates the vessels in the general circuit and contracts those in the pulmonary. In one such case demonstrated to the students in my wards at Bellevue Hospital the edema cleared up almost instantly after the administration of nitroglycerin—a very dramatic therapeutic procedure.

Venesection, too, is indicated and the withdrawal of a pint of blood, more or less, from plethoric individuals is sometimes helpful. If the patient is not digitalized ½ mg. of strophanthin (gr. 1/120) or 1½ mg. (gr. 1/40) of digitoxin may be given into the vein or an equivalent dosage of digitalis, gr. v to vii (0.33 to 0.5 gm.). If digitoxin is used, take it up in a little alcohol and dilute ten times with water; it is insoluble in water. Atropin sulphate has been used; if used, give intravenously or intramuscularly in goodly doses, gr. 1/50 to gr. 1/33 (0.0012 to 0.0018 gm.).

Cupping, to be well applied, has the disadvantage of disturbing the patient; nevertheless, it can be attempted on the portions of the chest accessible.

Oxygen inhalations, fairly concentrated, may be attempted; but the procedure may be distressing to the patient and then should be promptly discontinued. Morphin sulphate, gr. ⅛ to gr. ¼ (0.008 to 0.016 gm.), intramuscularly quiets the patient, lessens his restlessness and diminishes his perturbation and thus lessens the burden on the heart and is a valuable measure. Hearts which have so failed need assistance and moderate doses of digitalis continuously seem to me to have lessened the frequency of attacks.

Pulmonary Infarcts.—A pulmonary infarct may occur in the course of hyperpiesia. Personally I have never seen such a case. In a slowly failing heart an embolus may be derived from the right heart or from the thrombus forming in the pulmonary vessels. A large infarct may cause sudden death, pallor or cyanosis, great oppression in the chest, dyspnea, syncope, feeble pulse and death. In smaller infarcts, pain, cough; in something less than half the cases bloody sputum. Pain may be referred to chest or to upper abdomen if the diaphragmatic surface is involved. Later follow friction rubs over the site of the infarct, symptoms of a localized consolidation or the involvement of a whole lobe with signs of fibrinous pleurisy or effusion. The attack may be initiated by a chill and there may be a little temperature even in a non-infected infarct.

The treatment is to secure rest and relieve anxiety by a hypodermic of morphin, and then to endeavor to establish some degree of compensation by rest and digitalis.

Angina Pectoris

Cardiac pain may occur in a healthy heart when it is put to it to meet a demand beyond its powers. In this instance, the pain must be expressive of exhaustion of the heart muscle and induced by reflexes through those spinal cord centers where the sympathetic nerves of the heart have origin and whose stimuli flow over the adjacent centers of the sensory nerves supplying the skin, subcutaneous tissues, and muscles. It is logical to conclude, and the belief is firmly held by excellent clinicians, that conditions of myocardial weakness may more rapidly express themselves in the same terms when called on for too great an effort. Hence, cardiac pain is an expression of an incompetent heart muscle; and yet no pain may be provoked in a weak heart, regardless of the strain put upon it.

In the second place, the heart pain is singularly associated with profound changes in the aorta and is ascribed by thoughtful students of the subject to involvement of the nerve plexuses about the root of the aorta; and yet again, pain may be absent in advanced aortitis and coronary artery involvement. We are impressed with the feeling that both explanations of cardiac pain are acceptable in certain cases. Finally, it may be possible that toxic influences affecting these nerves or overflowing upon these centers from organs other than the heart may give rise to pain in the cardiac distribution.

This is not the place to discuss at length theories of the cause of pain: intermittent claudication of coronary arteries, stretching of heart muscle, spasm of heart muscle, and so on; but it is the place to emphasize that from a practical standpoint the pain means cardiac distress and is an appeal, ranging from a hint to a stormy protestation, for rest.

It is true that in most fatal attacks of angina pectoris the coronaries are found diseased; but this finding is not invariable and many patients who, having had attacks of angina, die of something else, show no signs of coronary disease, and extensive coronary disease is repeatedly found in hearts that were never the seat of angina. There may be, too, a singular lack of relationship between the severity of the attacks and the fatal issue. The pain itself is of more or less significance depending on concomitant factors. Age is one of these factors. Men and women in the fifties and sixties are far more likely to entertain a serious angina than earlier in life.

I have come to be singularly suspicious of people of this age with submanubrial or high substernal pain with radiation into the arm or arms provoked by effort, especially on taking an incline or facing a wind, and who have normal blood-pressure and a paucity of physical findings. There are curious facts about the exercise that provokes these pains. Many who can walk up stairs with impunity suffer an attack on walking up an incline; again and again patients attest to their onset on starting out for a walk; are obliged to stop until the pain passes; suffer recurrences two or three times, then go on for any distance without further trouble; such was the history of a man of sixty years, who stopped three times in as many blocks by pain, finished the day duck shooting, with a good bag and no discomfort. He died in an attack within the week following.

Syphilis with its accompaniment of aortitis, or if found in the history even when definite physical signs cannot be made out, gives an unfavorable prognosis for the anginal attacks. On the other hand, attacks in women at the time of menopause are known to be far less serious and must be assumed to accompany a different pathology. Prolonged mental and physical fatigue associated with anginal pains and more especially in those removed from anginal age and the victims of toxemic infections or tobacco or coffee habits offer a more favorable cardiac prognosis.

I am convinced that in many cases of angina pectoris, whether the pressure be high or low, toxic factors have a rôle in inducing attacks; that foci of infection, obvious or latent, can afford a steady, long-continued, deleterious effect on the myocardium, inducing fatigue on lesser efforts than would otherwise provoke an attack and perhaps affect nerves and nerve plexuses directly. Certainly such foci should be considered, searched for and, if found, appropriately treated.

The cardiac pains accompanying hypertension with which we are more immediately concerned seem to me to offer a special group. In the first place, they are commonly accompanied by an aortitis more severe in type than in the low blood-pressure cases and in so far offer the conditions for angina pectoris assumed by Allbutt; in the second place, involvement of coronaries inducing myocardial degeneration and a never-ending labor against abnormal resistance offer the factors for muscular fatigue that appeal to Mackenzie as an adequate explanation for the attacks.

The impression I have gained from these cases is that the attacks are more nagging, long continued, and more likely to occur at rest as well as during effort than in the normal or low blood-pressure cases.

It does seem in many of these cases that we were dealing as with a sensitive tooth, that every whiff of air sets off into a grumble or a sharp toothache. I think I can distinguish in this type a group of women, at or not far removed from menopause, in whom the nagging is incessant. These patients have much hyperesthesia of the skin, subcutaneous tissues and muscles. They look in fairly good physical condition and impress you as suffering from a severe neuralgia rather than from a storm that threatens their existence. The attacks do not simulate the classical attacks of angina major; neither are there striking evidences of cardiac decompensation. I have seen none of these die in the attack.

Among the men, too, the cardiac pains are very persistent; sometimes when at rest; but they are provoked or aggravated by effort and often slight effort; they are prone to continue for a long time, but at last there are increasing evidences of cardiac inefficiency and death in an attack or from cardiac failure.

Finally, in some of the hypertensives the attacks begin with severity, take on more of the classical character, are accompanied by rapid cardiac inefficiency and the patient dies suddenly in the attack.

Angina major, with its agonizing pain, nailing the patient to the spot in which he stands, making him fear to draw a breath, with a horrible apprehension of impending death, the so-called angor animi, must be very rare. I can scarcely recall a case among my hypertensives.

Of most significance are the pains or rather more often sense of constriction, like a hand grasping, felt at about the junction of the manubrium and gladiolus; this is of all degrees of severity and remains localized or radiates out to the upper extremities, the inner side of the arm and forearm, or to the shoulder or outer side of the arm; the location and extent of pain felt depending on the nerve distribution from the thoracic and cervical segments on to which the stimuli from heart and aorta overflow. Most commonly it radiates to the left arm, often enough to both; less commonly to the right only; exceptionally to the lower extremities, not rarely to the neck, grasping the throat, or up to the jaws or out to the tip of the mastoid or even more remotely. Less often the pain is felt over the true precordium and a pain at about the apex of the heart, especially in women, is commonly enough dissociated from any true cardiac disease. Numbness and paresthesia, pareses or cramps, may accompany the pain.

The sympathetic system shares more or less extensively in the attack: vasomotor disturbances, flushing or pallor, polyuria, sweating, salivation, belching.

Sometimes the pain is felt in the epigastrium and is spoken of as *abdominal angina.* It radiates to the back or up the sternum and out the arms. It may be of agonizing intensity and be mistaken for gastric perforation. It may happen that pain is felt in the arms for long periods, years before a typical anginal attack appears. Mackenzie has spoken of the pain as a reflex protective function; that is, it forces the patient to an immobility that lessens the demand on the heart; it is well put.

Treatment of Angina.—In those cases of angina in which the pain is provoked by effort and excitement there is no question that rest of body and mind is indicated. Such patients should be put to bed or at the most allowed to lounge about on a couch or easy chair and maintained in the attitude in which they are most comfortable. Rest for two, three or more weeks, or until attacks that come on even at rest shall have disappeared, should be insisted upon. During this period mild sedatives to the nervous system to decrease restlessness and anxiety and induce sleep should be given; for example, bromids of ammonium or the mixed bromids of sodium, potassium and ammonium, gr. xv (1 gm.) three and four times a day. The bromids may be replaced gradually by luminal in ½ gr. (0.030-gm.) doses. During this period mental rest is as important as physical, and sources of worry, fatigue, or excitement, long visits from boresome people, and consideration of business details, must be eliminated.

Insomnia is a potent source of fatigue and invites attacks. Courses of bromids and luminal, as advised, prepare the way for sleep; if they prove inefficient, mild hypnotics may be used. My favorite is trional; the usual dose is gr. x to xv (0.66 to 1 gm.) and it may be repeated in two hours; but often I find gr. v (0.33 gm.) is enough; another way to administer it, excellent in the nervous type, is to give it during the day at two-hour intervals in doses of gr. ii (0.12 gm.). I know of no advantage offered by sulphonal. Chloralamid in gr. xx to xxx (1.33 to 2 gm.) doses, washed down with a little cold water (not in a hot menstruum which breaks it up) or dissolved in an alcoholic menstruum may be used. Another mild hypnotic is adalin in gr. v (0.33 gm.) doses; more potent and more toxic is veronal or medinal in gr. v to viiss (0.33 to 0.5 gm.) doses. In rebellious cases (hyperpietics), chloral hydrate, in amounts of gr. x to xv (0.66 to 0.5 gm.), well diluted in water, is indicated and when the pain is responsible for insomnia, morphin, gr. 1/8 to gr. 1/4 (0.008 to 0.016 gm.). This latter should be used with great caution in the nagging types of angina in women at menopause as habit is easily established. The diet should be light during this rest period and the bowels regulated.

Digitalis.—There is not a general agreement about the use of digitalis in these cases; many clinicians have noted that as the compensation is established the anginal pains increase, just as they have noted the disappearance of anginal attacks with cardiac failure. This is being placed between the devil and the deep sea, indeed. My own experience with the drug has been happier. I use it as a "tonic" or "food" in small quantities of gr. iss to gr. iii (0.1 to 0.2 gm.) a day, and have received the impression that it has been helpful in cases of myocardial exhaustion or inefficiency, but that in hearts of powerful beat and in certain women at menopause it does no good, and perhaps aggravates; but "experience is fallacious and judgment difficult" and each case must be studied individually.

After a period of rest of weeks, depending on results, the patient is allowed about. If all has gone well and the pain has disappeared this is allowed in three to six weeks; then, as after the restoration of any break in compensation, the heart's improvement is tried out; the patient is permitted to walk on a level, slowly; gradually increasing inclines are attempted until recurring pain calls a halt or the heart demonstrates that it can meet ordinary demands. If the pains do not disappear entirely, we cannot contemplate keeping the patient permanently bedridden; it is exceedingly bad for his morale. Increasing freedom is allowed, studying the kind of effort and the kind of environment that provokes the attacks in an effort to establish its limitations and teach the patient how to live within them.

I have dwelt upon this preliminary effort to put the heart on a higher level of efficiency because of the gratifying results that have been obtained. I have watched patients who could not walk a block without pain after such a rest period resume their normal habits with no recurrence for years, provided deliberation and discretion were exercised. Such hearts have entered into a new pact with their owners—they will not stand for violations of the agreement.

Treatment of Acute Angina.—When once the physician has been summoned to attend a case of angina pectoris, he should instruct the patient what to do in an emergency and thus save him the anguish of waiting the physician's coming.

Nitrites.—Lauder Brunton certainly conferred a boon on humanity when he introduced the nitrites in the treatment of anginal attacks. The form in which the nitrites are usually administered or self-administered is by inhalation of the highly diffusible amyl nitrite; this is commonly put up in small, readily crushed, glass ampules or "pearls," containing ♏ iii to v (0.2 to 0.33 c.c.). Placed between the folds of a handkerchief, napkin or towel, the "pearl" may be crushed between the fingers or by a light blow by some hard object and the fumes quickly inhaled (to retain the crushed fragments of glass they are sometimes inclosed in a meshed fabric). The effects are instantaneous and almost miraculous. A flushing of the face, intense in character, a throbbing of the head and a sudden relief of the pain is what happens.

Nitroglycerin.—Almost as prompt is the effect of nitroglycerin when properly administered. It may be given in doses of gr. 1/100 to gr. 1/25 (0.0006 to 0.002 gm.) or more, as spirits of glonoin (spiritus glycerylis nitratis), each minim containing gr. 1/100 nitroglycerin (1 per cent sol.), or as a tablet, preferably a fresh, readily soluble hypodermic tablet. I prefer to give them under the tongue and let them dissolve and absorb there. The effect is prompter than when given hypodermically and much more so than when swallowed. I do not know the toxic dose of nitroglycerin. I have seen it given in amazing quantities, but in most individuals the dose is limited by the frightful hammering headache which an excess provokes.

The rapid relief was at first attributed to a fall of pressure; and while in many instances the pressure does fall, this is not true in all cases; in some it has been observed to increase, but the results are equally good, and, moreover, it is as effectual in the cases of angina with low blood-pressures.

In attacks of lesser severity administration of nitroglycerin by mouth is preferable to inhalations of amyl nitrite as the by-effects are less disagreeable. With nagging, frequent attacks, we may use the more slowly but longer acting sodium nitrite, gr. i to gr. ii (0.06 to 0.12 gm.) every two or three hours, or erythrol tetranitrate, gr. ¼ to gr. i (0.015 to 0.06 gm.) every four hours.

Morphin.—When the nitrites fail, as they sometimes do, the first drug we have recourse to is morphin and it must be given subcutaneously and in large doses, for example, gr. ¼ to gr. ⅓ (0.015 to 0.02 gm.), and, if necessary, in fifteen minutes give in addition one-half the preceding dose; results are what we are after and we must not be too closely bound by textbook dosage; severe pain breeds a large tolerance for morphin. These doses of morphin may well be combined with atropin sulphate in doses of gr. 1/100 to gr. 1/50 (0.0006 to 0.0012 gm.), which, through its action on vagus endings, lessens somewhat the danger of cardiac inhibition through the vagus.

Chloroform.—If nitrites fail and morphin is not at hand, inhalations of chloroform may be tried. Osler has said that he has never seen any dangerous effects from its use, even in weak hearts; yet there are those who feel a hesitancy in its use, especially in low blood-pressure cases and if symptoms of shock accompany the pain. The best way is for the patient to self-administer it, if he can, by using a wide-mouthed bottle containing a sponge on which the chloroform is dropped; as he becomes drowsy, the bottle will fall from his hand and prevent overdosage.

Chloral.—Chloral is not considered an anodyne, but Mackenzie finds it useful in relieving long attacks or preventing their recurrence; given at night in 10 to 15 gr. (0.66 to 1 gm.) doses it may induce sound sleep and block off a nocturnal attack, or it may be given in doses of 5 gr. (0.33 gm.) four times a day to ward off the frequently recurring attacks.

O'Hare has warned us of the danger of coronary thrombosis and infarcts in the prolonged attacks of angina, that make active and heroic interference all the more imperative. Oxygen inhaled is said to afford some relief.

In lesser attacks (angina minor) hot drinks, water, or bicarbonate of soda, ʒi in a glass of hot water, or a simple carminative, like a half dozen drops of spirits of peppermint with the bicarbonate, or a little whisky or brandy in hot water may be helpful. A mustard plaster or leaf or hot fomentations to the precordium may prove of value.

If there are signs of circulatory collapse, strophanthin, gr. 1/240 (0.25 mg.), or 3 to 5 gr. (0.2 to 0.33 gm.) of digitalis in soluble form should be put into the vein or, failing this, deep into a muscle. Less valuable are intramuscular injections of camphor in oil, gr. v (0.33 gm.), or the double salt of caffein and sodium benzoate or salicylate, gr. v to viiss (0.33 to 0.5 gm.). Aromatic spirits of ammonia may be administered by mouth or the stronger water cautiously inhaled.

General Treatment of Angina.—The general treatment is that laid down for essential hypertension but with added precautions. Excitement, worry, excessive physical effort or hurrying, excessive mental work all invite recurrences and must be avoided. As has been said, taking inclines, walking in the face of the wind, especially cold wind, are peculiarly apt to provoke an attack. Cold air, chilling the skin, and perhaps acting reflexly by its irritation of the nasal mucous membrane, may precipitate an attack. The warning about proper clothing in hyperpiesia should be more emphatically made in the anginal cases and they should be especially instructed to put on a warm outer garment no matter how brief an exposure to very cold air.

Such patients should not be confined to bed but we can insist on their carrying out advice as to shorter hours of work, more rest and more vacations. We give more careful attention to any insomnia, using bromids, luminal, trional, adalin as described.

Foci of infection should be diligently sought for, in sinuses, teeth, tonsils, middle ears, genito-urinary tract, gastro-intestinal tract, respiratory tree and measures taken to eliminate or treat such foci; the organisms determined, vaccines made and a careful administration considered.

Diet.—The dietetic restrictions have been dwelt upon, but one may add that alcohol, tea and coffee had better be eliminated, unless careful observation convinces that they play no rôle in inviting the attacks, but even then they were better restricted. The same may be said of tobacco.

If there is much flatulency one should suspect carbohydrates; at any rate the stools should be studied. One patient, who had suffered for months with cruelly recurring attacks day and night, got almost complete relief for six months or more when his salt intake was restricted; and this case was not a hypertensive. It is well in these cases not to indulge in large meals and especially at the end of the day; perhaps it is safer to have the heartiest meal at midday, if suitable rest may be taken after the meal. It is difficult to explain the relation between the eructations and flatulency so common in these cases and the cardiac condition. Such manifestations are peculiarly associated with the anginal attacks and with lesser cardiac pains. It has seemed to me that the gastro-intestinal disturbances and the cardiac pains were both expressions of the same cause operating on sympathetic and parasympathetic systems or that irritation beginning in one territory was reflected to the other.

Personally, I am unhappy when my cardiac cases have much collection of gas in stomach and bowels. The quantity of gas expelled by some of these patients is colossal. Its very amount makes fermentation an impossible cause. Aërophagy, I am sure, plays a rôle in some; they have gastric discomfort that they attribute to gas and in the effort to expel it swallow air; they are veritable cribbers; some collect such quantities so suddenly it seems as if their stomachs had reverted to air-bladders, excreting gases; sudden bloatings occur, such as one sees commonly in women that seem attributable to rapid dilatation of the intestinal walls as if all vagus motor influence was released. Relief of the gastric and intestinal disturbances should be prompt; one may give carminatives, such as spirits of peppermint in water, with or without soda [ʒi (4 gm.) in a glass] or Hoffman's anodyne, spirits of chloroform, compound spirits of lavender, aromatic spirits of ammonia or a combination. One may, and all the more if the patient is restless, give bromids as a sedative.

The bowels should receive attention. Constipation is to be avoided. I do not believe in drastic cathartics. Regulation of the food with proper consideration for roughage is best of all. Mineral oil, agar, bran, figs, prunes, grapes, dates are all helpful. Of drugs, the mildest cascara, aloin, compound licorice powder will answer the purpose. The patient should not strain at the stool.

Exercise.—This is best taken by walking on a level; later slight inclines, always leisurely and never disregarding any discomfort it may provoke.

Drugs.—As noted above, if the heart has shown decompensation, I like to give small doses of digitalis, gr. iss to iii (0.1 to 0.2 gm.), a day; but appreciate that there are some individuals who seem to have more discomfort during its administration; in such patients its use is contraindicated.

Atropin.—Allbutt advocates its use emphatically. He believes that one form of death is an "inhibitive shock" and that the well-known action of atropin on the vagus protects the heart against this accident. He uses it systematically and in increasing doses as tolerance is established. I have used it as he advises and have felt that in some cases it had a distinctly ameliorative effect on the number and severity of the attacks, in addition to whatever protective action it might exert through its action on the vagus. In minor attacks one tries many measures of relief; but it is hard to convince oneself of their efficacy. I have seemed at times to get results with suprarenal preparations (I have used adrenal residue of Arlington Chemical Company 25 per cent and 50 per cent in doses of gr. i every two to four hours); again with luminal in gr. ss (0.030 gm.) doses three or four times a day. I have had no success with benzyl benzoate. Arsenic has had some repute. I have had no experience with it. Inhalations of oxygen for short periods two or three times a day and high frequency current are measures regarding which I am unable to say anything definite.

Piersol in a study of 160 cases of hypertension, including nephritics, noted 18.3 per cent of anginas, of whom only 3.7 per cent died of angina. In my first series of 50 cases of essential hypertension I noted 12 per cent of anginas, of whom only 1 died of an attack.

Coronary Thrombosis and Embolism

Coronary thrombosis and embolism, the former more common, are accidents that may befall the heart in essential hypertension. Of course, the arteriosclerotic changes in the coronaries favor thrombosis, and bland emboli, formed in a weakening heart, especially a fibrillating one, favor embolism. Stoppage of a main branch may be instantly fatal; on the other hand, lesser infarcts may occur without symptoms. The clinical expressions of the non-fatal cases are precordial pain, dyspnea, cyanosis, oppression, apprehension, and at times edema of the lungs. There may be gastric symptoms. The heart is rapid and markedly irregular, or a paroxysm of auricular fibrillation may ensue. Areas of hyperesthesia may develop over Head zones. Cardiac decompensation may rapidly follow and death occur in twenty-four hours, or set in more slowly in a few days or weeks; or the patient may quite recover from a distressing attack and with care survive for months or years. Blocking of the vessels may and often does give rise to fatty changes and gradual weakening of the heart muscles, and cardiac aneurysm may follow.

Treatment.—Treatment consists only of rest and morphin hypodermically to insure the rest, physical and mental, and the decompensation met by a beginning dose of strophanthin into the muscle (½ mg. of crystallin strophanthin), followed by digitalis, gr. vi to x (0.4 to 0.66 gm.) a day for three days; further dosage to be determined by results.[1]

High Blood-pressure Stasis

As has been said, one of the striking features of the heart's conduct in essential hypertension is its heroic struggle to the last gasp, maintaining or associated with the maintenance of a high pressure and a curiously persistent high diastolic pressure almost to the last.

Under such circumstances, it is easy enough to underrate the crying need of the myocardium for rest and assistance. Arthur Elliott has well described this condition. He considers those evidences of myocardial failure touched upon earlier in the article, rapid pulse, gallop rhythm and pulsus alternans as peculiarly associated with the heart of high-pressure stasis. I have already expressed my opinion of the significance of a gallop rhythm and the ominous import of a pulsus alternans. I have also dwelt on the serious significance of the attacks of cardiac asthma associated with this kind of a heart; attacks that wake a patient out of sleep, soon after falling asleep, and compel him to sit up to get his breath. Cheyne-Stokes' respiration may occur which affords a gloomy prognosis, though patients may rally from that condition. The left heart seems to bear the onus and it would seem as if the right heart remained relatively good; this results in a heaping up in the pulmonary circuit; hence, the râles of congestion appearing early at the bases and the attacks of pulmonary edema later. However, the right heart does eventually feel the weight of the back pressure and in turn throws back on the venous system, resulting in an enlarged and tender liver and gastro-intestinal dysfunction as an expression of stasis in the portal circuit and an albuminuria as the evidence of stasis in the renal circuit; but is not accompanied by edema and collections in the serous sacs until very late or in exceptional instances.

Treatment.—The immediate and most urgent need in these cases is rest. It is criminal to allow these cases to keep about while we dally with drugs. They should be put at rest in the most comfortable position with a restricted intake of food and of fluids. Venesection is indicated in these cases and all the more so if an hypertensive crisis, an episodic increase in blood-pressure, has occurred. The amount taken may be from 300 to 900 c.c. (10 to 30 oz.), depending on the size and plethora of the patient and the urgency of his symptoms. Pulmonary edema intervening calls also for this means of relief.

---

[1] There are competent clinicians who take exception to the use of digitalis bodies in these cases.—Editor.

Digitalis.—This is an important paragraph for those who still have a dread of the constricting effects of digitalis on the vessels. This fear may be dispelled. The effect of the drug on the heart is only beneficial and it should be used fearlessly, in full dosage. If we fear to push it to the production of nausea and vomiting, as the slowing of the heart may not occur, or certainly occurs in a lesser measure than in fibrillating hearts, Eggleston's rule of 2 minims of tincture per pound of patient (amounting to 30 to 35 gr.) administered in three to four days will be very conservative; in thirty-six hours to two days in more urgent cases; and then such doses as the patient will eliminate each day, 1½ to 2 gr. Allbutt advises using the drug when the diastolic pressure rises and the systolic decreases, and this is excellent advice; though he speaks for digitalis less emphatically than he is prone to champion any method that appeals to him. What one usually sees is a fall of blood-pressure. I am accustomed to teach that digitalis brings the pressure nearer its optimun, raising it in those cases that fall with a failing heart and lowering it in these cases of high blood-pressure stasis. It is what one would expect with an improved circulation. Like every other case of decompensation, when the heart's forces have been restored so far as possible the patient should be allowed up and about gradually, the results of effort being watched.

We are warned by some writers against the use of digitalis when angina obtains. Certainly if it seems to come on or increase under the digitalis, the patient being at rest, it is wise to stop it. Such accidents as pulmonary edema, cardiac asthma, angina have been discussed elsewhere.

Auricular Fibrillation

It has been said by excellent observers that auricular fibrillation is rare in these cases, and so it is compared with its frequency in the so-called chronic myocarditis or, to avoid pathological connotation, the senile heart; but while the percentage is small one sees no inconsiderable number if he treats many hyperpietics.

Auricular fibrillation is always a serious condition, for now the heart which still has to drive against the great resistance ahead wastes a great deal of its energy by perfectly useless beats; for this is a condition in which the irritability of the auricular musculature has become such that it has overridden the pacemaker, the regulator, and asserted its primitive prerogative of initiating a heart beat, but in a disorderly manner. Four hundred or more beats of the auricle initiated per minute result in a fibrillating movement of its fibers without coördinating to contract the auricle as a whole; these impulses sweep down upon the bridge of His which permits the crossing of such numbers as it can, 140 to 180 per minute, but in an absolutely irregular, arhythmic manner. Some impulses initiate the ventricular contraction before it contains enough blood to forward appreciatively the movement of the blood as a whole. The contraction goes for naught and fatigue results. In these cases the right heart shares fatigue with the left and throws back on the great veins; the liver becomes congested, the portal system is engorged, ascites and hydrothorax occur. Perhaps this is the reason why one rarely sees the sudden issues of pulmonary edema and the anginas in these cases. The left heart when staggering between the drive of a competent right heart behind and contracted arteries ahead falls like a spent runner at the tape; but with a weakening right heart is relieved of its drive.

Treatment.—With the onset of fibrillation there is rapid cardiac decompensation such as one sees in the same condition in senile hearts and in mitral stenosis; but this is the condition *par excellence* amenable to digitalis therapy. Undoubtedly if Withering had not noted its remarkable results in just these cases, we should not have come to know digitalis, or at least its use would have been delayed. Without discussing the minutiæ of digitalis action, it may be said that it does exactly the things we want it to do: it slows the heart, both through its stimulating action on the vagus nerve and by its depressing action on the bridge of His, so that fewer auricular impulses get across the bridge to excite the ventricle to contraction. This means a great economy in effort and time for most of the diastoles to fill the ventricle before the systole is provoked to discharge it, and, secondly, it energizes the individual heart beat, makes it more powerful. These cases then are treated like auricular fibrillation in any other heart, by rest in the most comfortable position, and that in severe cases is an upright one, morphin for sleep by hypodermic, gr. 1/4 (0.015 gm.), restricted diet and fluid; a strict Karell diet of 800 c.c. of milk for the first three or four days.

Digitalis.—Digitalis is given to the point of digitalization as evidenced by the desired therapeutic effects or by toxic manifestations. In an urgent case one may get in 30 gr. (2 gm.) of digitalis in less than thirty-six hours, giving 15 gr. (1 gm.) at the first dose; half that amount in four hours and the rest in three or four doses at four-hour intervals. This is following Eggleston's rule of 2 minims per pound of patient for a man of some 150 pounds. This dose, however, is materially added to if results are not obtained. If the administration is drawn out to four or five days, it often requires 50 or more grains to get the desired effect. It may be given as the leaf, as tincture, or as infusion. One must be assured of a drug of high assay; because most practitioners cannot be so assured, one may well prescribe some of the solid or liquid preparations of high assay put out by reliable houses under different trade names.[2] Treatment may be begun with an intramuscular dose of strophanthin, which is eliminated for the most part in thirty-six hours, 1½ mg. (gr. 1/40) of the official amorphous strophanthin [equals roughly 5 gr. (0.33 gm.) digitalis] or better ½ mg. crystalline strophanthin (ouabain) equals roughly 6 or 7 gr. (0.4 to 0.5 gm.) digitalis. The oral administration of digitalis can follow at once, but if digitalis has been already given, strophanthin should be used cautiously, with a full knowledge of how much digitalis has been given and how much digitalis the dose of strophanthin is equal to. Its use by vein under such circumstances should be deprecated. Strophanthus orally administered should be decried. It is poorly and irregularly absorbed and consequently poorly controlled.

One should see as the result of satisfactory digitalis dosage a slowing of the *heart beat;* the deficit between heart beat and pulse rate should approach zero. If the pulse drops to the sixties, the drug should be stopped. Diuresis should also occur and edemas disappear. If diuresis is tardy, theocin for three doses of 5 gr. (0.33 gm.) each, or diuretin for two or three days in doses of 7½ gr. (0.5 gm.) three times a day may initiate the flow of urine. Improvement in well-being, sleep, appetite and breathing should accompany this.

Toxic accumulation is shown by nausea, vomiting, diarrhea, very rarely by mental disturbances, and still more rarely by disturbances of vision with intense exaggeration of white light coupled beats (supraventricular beats and premature ventricular systoles coming in pairs, with longer intervals between the pairs) or heart-block, as shown by slow, regular beat thirty-five to fifty, sometimes more. A fibrillating heart, while still fibrillating, can become regular only by a block at the bridge of His, which cuts off all impulses rising from the auricle, but often when the heart slows to the fifties or sixties a careless observer thinks the pulse is regular when it is not. A little effort on the part of the patient which quickens the pulse brings out the arhythmia more distinctly.

Cardiograms show the coupling, the blocking and inversion of T-waves. Such accumulations call for interruption of the drug.

For methods dealing with accumulation of fluids, see Cardiac Failures with Edema.

Cardiac Failures with Edema

A certain number of hearts gradually weaken, the left ventricle throwing back the burden on the right heart with the result of a general venous stasis such as is seen in fibrillating hearts and in the valvular hearts of rheumatism; anasarca, ascites and collections of fluid in other serous cavities ensue; the patient becomes water-logged and orthopneic. It would seem that in some instances this form of failure is associated with an infection, and the occurrence of fever, hemorrhagic eruptions, pleurisy, pericarditis, and meningitis would seem to warrant the supposition. In such infections, a rather rapid fall of blood-pressure is noted.

---

[2] With no expression of preference, I will name digitan, digiglucin, digitora, digifolin, digipotin.

Treatment.—This has been described in the consideration of the fibrillating heart.

Rest.—Rest in the most comfortable position is indicated.

Sleep.—Sleep should be induced by the use of the milder hypnotics, such as bromids, adalin, trional, chloralamid, and in the urgent cases, orthopneic and worn out with insomnia, morphin hypodermically, gr. 1/4 (0.015 gm.) and repeat with half the dose.

Diet.—Diet is sharply limited, especially in fluids. It is best to begin with a strict Karell diet of 800 c.c. of milk in 200 c.c. quantities and add no more fluid than relieves an urgent thirst and then after a few days adding more articles, but keeping the diet dry. Salt should be rigorously eliminated.

Digitalis.—Digitalis is used in full doses (see Auricular Fibrillation) and the use of Eggleston's formula for digitalization of two minims per pound of patient may be taken as a guide. It must be remembered that the rule holds only for averages and that more may be required and some patients may respond to less. The results are not as satisfactory as in the fibrillating heart; first, because the efficacy of the drug in the latter is due largely to the reduction in the total number of beats through blocking at the bridge of His by the drug; while in the cases under consideration, the reduction in number of beats depends on the action of the drug on the vagus. In some cases the heart may be materially slowed, some to the induction of a block; but this is not constant and the slowing may be very slight or not occur at all; in the second place, fibrillation may occur with a muscle still capable of performing its functions if spared the excessive and unnecessary beats; but in the gradual failures under discussion, the heart is demonstrating its incapacity at even a normal or moderately accelerated rate; in other words, the myocardium in these cases is usually bad. However, even if the heart is not materially slowed, the drug may still be of value in energizing the individual beat. The evidences of a toxic dose in these cases are nausea and vomiting or diarrhea; increasing frequency of premature systoles, slowing of the beat to the sixties; this, of course, calls for an intermission of the dosage and on disappearance of toxic symptoms, beginning with a dose that the patient can eliminate, 1½ to 2 gr. a day.

One of the earliest evidences of the digitalis action is diuresis; if, when the digitalis has been administered to or near to a full dose, diuresis has not appeared, it may often be initiated by the administration of a diuretic of the purin derivatives. One may use theocin in 5-gr. (0.33-gm.) doses three times for one day; longer usage may cause renal irritation; another drug of the same series is the double salt of theobromin and sodium salicylate in doses of 7½ gr. (0.5 gm.) three times a day for two or three days; longer administration may cause renal irritation and damage. Caffein sodium benzoate in doses of 5 gr. (0.33 gm.) has a similar diuretic effect, but its tendency to aggravate insomnia makes it less desirable than its congeners mentioned.

Removal of Fluid.—Diuresis may first begin when relief is afforded the tissues. The introduction of Southey's tubes on either side of the ankle just below the malleoli, or scarification of the edematous tissues of the leg over its lower half or third by stabs with a bistoury guarded with adhesive to one-quarter inch of its point, or by cross hatching with the edge of a scalpel, will release an amazing amount of fluid, which seems, by relieving the tension in the tissues, to facilitate absorption into the blood-stream and elimination.

The legs are kept in a dependent position, the feet in a foot-bath or other receptacle containing a bland antiseptic fluid, like boric acid solution. This is of course easy when the patient is sitting up. When the patient is recumbent, the knees may be bent and the feet kept in the foot-tub; but this attitude is tiring and must frequently be relieved; moreover, the fluids in the thighs follow gravity and collect about the upper portion of the thighs and dependent tissues of the back. The scarified areas may be allowed to drain into loosely applied aseptic gauze. It must be emphasized that this procedure is essentially surgical and that every rule applicable to surgery is indicated; a slip in technic can readily result in infection of these highly susceptible tissues and infection in such cases is a very real danger to the myocardium.

Thoracentesis.—Thoracentesis should be practiced when the collection in the pleural sacs is an obvious factor in respiratory embarrassment. The usual technic is observed with a Potain apparatus or vacuum bottle. The fluid should be removed slowly to avoid a possible edema in the compressed lung, which may occur if the pressure of the fluid is too suddenly removed. Cough or distress of any kind should call a halt in the procedure. The removal of moderate amounts is often followed by rapid absorption.

Ascites.—Huge collections may be removed by tapping; this is but rarely necessary; if the circulation is relieved by removal of fluids elsewhere as described above and diuresis is established, the ascitic fluid will be rapidly absorbed; if, however, diuresis is not established, the removal of a part of the ascitic fluid may bring about the desired result.

Hydropericardium.—Even less frequently is it necessary to tap the pericardium, but where it is evident that a huge collection of fluid is present and is a source of embarrassment to the circulation, its removal should follow. This embarrassment is shown by much dyspnea, with symptoms of pressure, cyanosis of face, distended veins in the neck, cough and perhaps loss of voice and difficulty of swallowing. The best place to enter is halfway between the left border of the cardiac dulness and the palpable apex, or in large collections through the left posterior chest as in hydrothorax.

Cardiac Murmurs

Cardiac murmurs are heard commonly enough with the heart of hypertension; they are nearly always systolic in time, and heard at the apex or at the base. Those at the apex may be ascribed to a relative insufficiency of the mitral valve or to a sclerotic change in the valve. In certain systolic apical murmurs, one can trace them to the aortic valve, where a murmur of the same quality and pitch can be identified at its maximum intensity. It is my belief that in the case of most soft systolic murmurs at the apex, a conjecture as to the pathology of the valve remains a mere guess. The systolic at the base, loudest over the first or second intercostal spaces adjacent to the sternum, may be ascribed to pathological changes in the aorta (dilatation, roughening).

I am surprised again and again when detecting these murmurs in patients whom I have not seen for a long time to find that these murmurs have been recorded years before; so that they seem fairly constant after being established and in themselves have ceased to sound a note of alarm. Of the diastolic murmur and its significance I cannot speak with conviction. A certain number of these hyperpietics who have developed a diastolic murmur are luetic and this infection always puts a heavy black mark against the prognosis. Of the non-luetic I cannot recall enough in my considerable series to have impressed me with either their frequency or seriousness; but I am aware that this "forcing" of the aortic valve is looked upon by some able clinicians as introducing a serious element into the case.[3]

Paroxysmal Tachycardia

Just what the relation of this condition to the hyperpietic state may be is not clear. That the hearts of hyperpiesia are irritable is shown by the frequency of premature systoles, and the occurrence of paroxysmal tachycardia may be attributable to such irritability. The occurrence is relatively rare and in some of my cases began in childhood, before hyperpiesia had, in all probability, manifested itself. I have seen the condition quite disappear in cases of marked hypertension after annoying recurrences for years. Most individual attacks stop of themselves after a short or long period of time and little harm is done by the experience, if the heart muscle is sound; but it is possible for the heart to fail in long attacks or when the musculature has been damaged.

---

[3] Relative insufficiency of the aortic valve is probably much more frequent than we have usually been led to suppose.—Editor.

Treatment.—Pressure on the vagus nerve in the neck often succeeds in interrupting the paroxysm. The relation of the vagus nerve to the carotid artery is such that if compression of the artery along the anterior border of the sternocleidomastoid muscle is made at about the junction of the upper with the middle third of its course, and against the underlying vertebral processes, the nerve is pinched and mechanically stimulated. When such pressure is made on the right side those fibers going to the pacemaker and the right auricle are stimulated, a cessation of heart beat for a brief period is elicited and the pacemaker is restored to its control. The onset and offset of the paroxysm are between two beats. Occasionally pressure over the left vagus is efficacious, perhaps in those cases in which the paroxysms begin in the junctional tissue to which the left vagus sends branches. Both vagi should not be simultaneously submitted to pressure. Ocular pressure, a steady pressure on the eyeball, usually the right, is often followed by a cessation of the paroxysm. It is rarely successful with a pressure less than one inducing pain, equal to some 30 mm. of mercury. This stimulus is conveyed eventually to the vagus.

Patients come to perform certain maneuvers in which the vagus is unconsciously stimulated to bring the paroxysm to a stop, such as taking deep breaths, swallowing water, especially with the neck on the stretch, vomiting, telescoping the neck into the shoulders until the face and neck are deeply congested, then standing and stretching. Some learn to compress the vagus as described above. Usually there is some eructation of gas following the attack. Sometimes patients stop the attack by belching gas; commonly a sense of gastric uneasiness precedes the attack.

Drugs.—The only drugs of value are the digitalis group, and in my experience the dose must be massive. I have succeeded in stopping several attacks by the use of colossal doses of strophanthin after the suggestion of my colleague, Dr. Walter Niles; but hesitate to recommend the quantities I have used, as occasional accidents have occurred in strophanthin medication. It would be safer no doubt to use huge doses of digitalis; but I doubt if quite the same success would follow though possibly they might if given by vein. For the failing heart of a paroxysm that cannot be interrupted, digitalis is indicated to the point of digitalization (some nausea, vomiting, diarrhea) or until the theoretical dosage for the weight had been administered. I have never seen bromids or morphin stop the attack; but theoretically bromids are indicated to lessen the irritability of the cardiac muscle and block off recurrences. Whether gastric disturbances induce their onset or not is a question; but the diet should be simple and designed to lessen flatulency.

PREMATURE SYSTOLES

It has been said that no man gets by thirty-five years without having experienced premature systoles. Certainly we see them commonly when no evidence of heart impairment can be determined; but we do look upon them as expressions of increased irritability of muscle substance, due to fatigue, nerve exhaustion, or toxic substances, such as tobacco, tea and coffee and toxins of infections. In the absence of disease they elicit little concern on the part of the practitioner, but in conditions in which it may be presumed that the heart has undergone organic change, such instability as premature systoles suggest a less satisfactory state of the myocardium than otherwise. These premature systoles are almost always of ventricular origin; if auricular, they have seemed to me to be more serious. They may occur after each supraventricular beat and simulate a pulsus alternans which bespeaks a much more serious myocardial state. The pause between the couples helps us to discriminate between the two conditions unless arising from the node; the cardiograms are distinctive. The premature systoles seem to occur with rising pressures or perhaps with increasing irritability due to lessening blood supply through damaged coronaries, possibly with states of mental excitation or prolonged stress.

Treatment.—Mackenzie has found some diminution of the number of premature systoles under nitrites. It has seemed to me that rest and sedatives, such as bromid, have had a steadying effect and that they recur less frequently as some degree of compensation is established under digitalis. It must not be forgotten, however, that accumulations of digitalis are often expressed by the occurrence or increase of premature systoles, the most exaggerated form of which is the coupled beat of digitalis.

VASCULAR ACCIDENTS

The three vascular circuits, involvement of which in arteriosclerosis entails the most serious consequences, are the cerebral, the coronary and the renal. The results of coronary sclerosis, such as angina pectoris, coronary thrombosis or infarct and cardiac failure have been discussed in the section on the Heart; the changes in the renal vessels and their sequelæ will be taken up later.

CEREBRAL VESSELS

Early changes in the cerebral vessels are hinted at more or less broadly by mental fatigue or lag; increasing effort to accomplish wonted mental tasks, slight forgetfulness of engagements, recent occurrences or names;

increasing irritability and restlessness, slight change in temper, impairment of judgment, insomnia, headaches, especially early morning headaches, occasional dizziness, fleeting aphasia or monoplegias or sensory disturbances, such as paresthesias and numbness, or even hemiplegias, more or less short-lived. These latter conditions have been variously referred to intermittent claudication of cerebral vessels; to spasm, which is open to question; to momentary kinking of the branches of the middle cerebral by the erection of the main trunk from its loose bed during hypertensive crises; to minute hemorrhages; to localized edemas, and to localized effects of toxic substances, after the nature of uremic poisons. Any one of these or any combination is likely to occur repeatedly before a major accident. A complete and permanent hemiplegia or a rapidly fatal hemorrhage may be the first announcement of cerebral involvement.

One not infrequently finds decided change in the retinal vessels, which enhances the probability of changes in the cerebral vessels in cases of questionable symptomatology.

When the early and insidious symptoms mentioned occur, the patient should be kept under close observation, the condition of the heart determined and renal functional tests carried out, to afford, if necessary, suitable cardiac support (already discussed), on the one hand and to determine, on the other, how far renal inadequacy may play a rôle in the picture presented.

I advise that a period of complete rest, absence from business and avoidance of sources of worry be afforded the patient, with a gradual return to restricted activities as improvement indicates. This class of patients may show improvement over long periods of time. It is well to study the pressure curves in these patients at such intervals as to appreciate in case of sudden accidents, if these be due to hypertensive crises.

Treatment.—The passing attacks of aphasia and monoplegia, etc., may be shown to be due to or accompanied by an increase in the blood-pressure and, if they are, treatment should be directed towards its diminution. The patient, under any circumstances, should be put to bed with the head slightly elevated, but not to so flex the neck as to obstruct venous return; a saline cathartic administered, a sharply restricted diet instituted, such as a Karell diet of 800 c.c. of milk per day in divided doses, with such an amount of water as will assuage thirst. Sedatives may be administered to diminish restlessness, such as bromids in 15-gr. doses (1-gm.) or, if restlessness and insomnia are decided, morphin sulphate hypodermically in doses of ⅛ to ¼ gr. (0.008 to 0.015 gm.). This, I think, is sufficient if there is no rise above the usual blood-pressure reading of the particular patient. If, however, there is a sharp rise, it is well to administer chloral hydrate, gr. v (0.33 gm.), with the bromids at four- or six-hour intervals. Nitroglycerin is indicated at two-hour intervals in doses of gr. 1/100 up to gr. 1/50 (0.0006 to 0.0012 gm.) or more.

Halt on the administration of the nitroglycerin is called by the violent, hammering headache or by attaining the desired fall in the blood-pressure. In place of nitroglycerin one may use erythrol tetranitrate in doses of gr. ½ (0.03 gm.) at four-hour intervals, or sodium nitrite in doses of gr. ii or iii (0.12 to 0.2 gm.) at the same intervals. In the cases with an increased hypertension phlebotomy is indicated. Twelve to 16 ounces (350 to 500 c.c.) may be withdrawn in plethoric individuals; in those with decided anemia I should hesitate to have recourse to this measure.

In the severe *apoplexies* not much more can be done than has just been mentioned; for example, rest, with head slightly elevated, no food during the early hours or day or two of unconsciousness, water carefully administered by mouth if it is swallowed, and if not by bowel, by small injections or by a Murphy drip; the retention of a quart a day will be sufficient. Care of the mouth, teeth, tongue, dead spaces between gums and cheeks, of pharynx and nose, with mild antiseptics applied by swabs or sprays, should be exercised.

Drastic cathartics do not appeal to me, but the bowel should be cleaned by enemata or irrigations. None of the sedatives mentioned are indicated during the period of unconsciousness unless restlessness obtains, when morphin may be used. Nitroglycerin or other nitrites are used to control excessive pressure and my experience has encouraged me to do a phlebotomy in the plethoric cases.

If convulsions repeat themselves after the onset, one may use morphin and do a lumbar puncture; if the fluid is under increased pressure, it may be allowed to escape very slowly until the increase in the pressure of the spinal fluid is materially diminished.

With return to consciousness, the feeding problem is more acute. Food should be given in small quantities, frequently repeated, of non-stimulating fluid, milk, or milk soups, purées of vegetables or rice or farina, soft toast, ice cream, eggs in moderation, cereals, thoroughly cooked vegetables, cooked fruit. The diet should observe moderation, low protein and low salt.

The nurse is more useful than the doctor in these cases; the care of the body, the skin, the genitals, the anus, the skillful posturing of the affected members, ventilation, quiet, all the niceties of her profession are indicated here.

Effort should be made early, but at first not too vigorously, to prevent contraction of muscles, and to keep them prepared for whatever restoration of brain function may occur.

To go into the many forms of cerebral disturbance incident upon vascular changes and accidents would be to encroach upon neurology, and the details of different aphasias, of cortical, subcortical, and basal lesions will not be attempted.

RETINAL VESSELS

The retinal vessels are involved in a considerable percentage of cases. It is the one and only territory in the body where the vessels lie bare to the view. The patchy nature of the arteriosclerosis is shown by them; for in one patient with a very high blood-pressure no changes in the retinal vessels may be seen at all, while in another with the same pressure, or one far less, the changes may be very pronounced. As one sees the advanced cases he feels impressed that there is a toxic irritant acting on the eye-grounds aside from changes induced by the pressure alone.

Early changes are seen in the displacement or flattening of veins at arterial crossings, by increased disproportion between the small arteries and large veins, later by the tortuosity of the vessels, by hemorrhages and by exudates. These latter are usually small, silvery in appearance rather than the massive, snow-bank, cumulus exudates of albuminuric retinitis, and there is little or no neuritis; and yet, this arteriosclerotic retinitis is repeatedly called albuminuric retinitis and chronic Bright's disease diagnosed as a result. How much the sight is affected depends on the distribution of the hemorrhages. The prognosis is worse in cases of arteriosclerotic retinitis than in those with normal eye-grounds, for the cerebral arteries seem more commonly involved in these; however, I have traced such cases for many years after observing these retinal hemorrhages.

THROMBOSIS

Mesenteric Thrombosis.—This may occur as the result of atheroma or by a narrowing of its opening from the aorta, or plugging by an extension of a thrombus formed on an atheromatous aorta at the origin of the artery. Hemorrhagic infarction of the intestine may follow, especially if the occlusion is effected rapidly. It occurs more frequently in the superior than in the inferior mesenteric.

The symptoms are severe colicky pain, tympanites and tenderness of the abdomen to palpation, vomiting, diarrhea and shock; the stools and vomitus may contain blood. The pulse is rapid and small; the patient is covered with cold perspiration, and the face is drawn; later constipation follows; sometimes a mass is made out, but usually the abdomen is enormously distended and symptoms of peritonitis dominate the picture. There may be perforation of the gut with peritonitis, as in a case of mine at the age of eight and a half years. Death follows in a day or two, or it may be delayed for one or two weeks. In some cases the symptoms are those of an intermittent claudication with severe colicky pain, distention, nausea, vomiting, eructations of gas and in one of my cases extreme apprehension of death. I have seen such a case go through repeated attacks of this kind with evidences of ulceration and yet rally to die suddenly six months later of cardiac failure.

Treatment.—For the acute cases only prompt surgical intervention can be of value. For the intermittent claudications, morphin, hypodermically, is most effectual; nitrites have been tried but are, as a rule, but little helpful. Hot cloths may be applied to the abdomen. Between the attacks I would advise against drastic cathartics, but would rely on warm, not hot, enemata.

Thrombosis of Peripheral Vessels.—Thrombosis of the lower extremities, occurring gradually, causes pain and often coldness of the part and cramps when the member is exercised; this is followed by relief when at rest. This form of thrombosis is the so-called intermittent claudication. In complete plugging, pain in the limb, severe and paroxysmal, follows, so severe that I have seen patients roused from a deep coma to cry out with it. The pulse cannot be felt below the stoppage; the part becomes white, then livid and mottled and gangrene ensues, usually dry, unless the vein is also thrombosed. How extensive this damage to tissue will be depends on the size of the vessel occluded and the collateral circulation of the part.

Treatment.—The part is slightly elevated, kept warm and, in case gangrene appears, hot, dry air is supplied. Gangrene, of course, is to be followed by amputation or excision.

In intermittent claudication excessive demands on the part should be avoided; warmth and gentle massage to widen the capillary bed and relax arteries are indicated.

Hemorrhages

Aside from cerebral and retinal hemorrhages already discussed, there are hemorrhages elsewhere to be considered. One of the most common is epistaxis. This may be the patient's first notification of any vascular disturbance. It is very prone to occur in the "plethoric" and is, in a sense, a safety valve, a natural phlebotomy, and the patient often feels the better for it; but it may be furious and demand interference.

Treatment.—The treatment that is most efficient is plugging the nostrils, bringing pressure to bear on the bleeding vessels which may be seen on the septum near the nasal orifice; but sometimes the bleeding vessel is not so obvious and the posterior nares must be plugged. This one may do in emergency by passing a small catheter through the nasal passages to the pharynx and, as it curves forward to the mouth, thread the eye with tampons with long strings attached and draw them into the posterior nares with the withdrawal of the catheter. This leaves one string through the nose to draw the plug into the nares and one string trailing from the mouth by which the plug may be withdrawn. Hemostatic tissue substances may be used on the tampon. If the patient is alarmed and restless, morphin may be used by hypodermic. If the bleeding occurs in the anterior part of the antrum, the patient may control it by pinching the alæ nasi against the septum.

Hemorrhages may occur from the stomach (hematemesis), or from the lungs (hemoptysis), even 2 or 3 ounces; or from the kidney, or elsewhere in the urinary tract (hematuria), or from the uterus, constituting metrorrhagia. One may rarely see little hemorrhages under the skin, and bloody spinal fluid indicates the occurrence of meningeal hemorrhages. Of the uterine hemorrhages, one must remember that fibroids are associated with high blood-pressure and that the metrorrhagia may be due to the fibroids. A vaginal examination should not be omitted.

This is not the place to discuss at length the high blood-pressure of cases of uterine fibroids. It is to be recalled that the age of hyperpiesia and of myomata uteri is the same and that the occurrence of the two may be merely coincidental not causal. Before drawing any conclusions we await a body of statistics showing the remedial effect on hyperpiesis of removal of fibroids.

Erythrocytosis

It is certain that in a goodly number of the cases of primary hypertension a blood count shows something more than the average number of red blood-cells; sometimes a rather striking increase. While, unfortunately, I have not done blood counts on all my cases, I have done a sufficient number to persuade me that some increase in red cells is a feature of the condition.

In Geisböck's disease we have a distinct polycythemia, from 6,000,000 to 11,000,000 red cells per c.mm. associated with a high blood-pressure without the enlarged spleen of that form of polycythemia called by the names of Vaquez and Osler, in which high blood-pressure is not a feature. In this syndrome of Geisböck's, renal involvements and cerebral hemorrhages occur, so that one wonders if a sharp line can be drawn between these conditions, for typical hyperpiesias with noticeable red cell increase shade into cases described as Geisböck's disease with a lower range of erythrocytes. There is no close parallel between the height of blood-pressure and the degree of erythremia, and the relation between the erythremia and blood mass or true plethora is open to discussion.

These patients are prone to epistaxes, headaches, dizziness and have the cyanotic hue of an overripe plum, the apoplectic facies.

Anemia

When anemia occurs in this class of cases, in whom the usual blood picture is one verging on erythremia, the prognosis is bad. Sometimes this change occurs with startling suddenness, the pallor increasing to a degree suggesting cachexia and resembling that of true Bright's disease. The pallid hue underlying patches or islands of the once florid countenance is very striking. It is not altogether clear in any one instance to what this may be due. When evidences of infection occur it is tempting to hold this responsible in some measure, but the patient's appearance suggests something more profound than a secondary anemia, except that of long-standing suppuration. Brown and Roth have recently discussed the anemia of chronic nephritis and their explanation may cover a certain number of cases of essential hypertension, especially those in whom the kidney has become seriously involved. While anemia may, of course, follow upon the severe hemorrhages that now and then occur in these cases, for example, an epistaxis, this is not the kind of anemia under discussion.

Brown and Roth quoted could not determine that hemolytic processes had anything to do with anemia, but thought defective blood formation played the important rôle; they call attention to the absence of regenerative efforts in the blood, the low percentage of reticulated red and nucleated cells, also the low platelet count. Polymorphonuclears, however, are not affected, so that it is not the bone-marrow as a whole, but its erythrogenetic function only that is implicated.

Endeavoring to discover the cause, they felt that diet could be excluded, but that some unknown toxic agent was operative, and acting concomitantly with evidences of renal decompensation, such as retained urea and creatinin. They consider the depression of the erythropoietic function but a local expression of a widespread action of this toxin, which affects the heart, the vessels, the kidney, and the retina. If kidney decompensation is made good, still the restoration of the bone marrow lags and with repeated decompensations the anemia deepens and takes on certain characteristics of the aplastic type. There is much about this explanation of the anemias of hyperpiesia that is appealing to me.

It is difficult to see what can be effected by treatment of the anemia as such until we know something of the cause. Bone marrow may respond to the arsenicals, but the arsenicals do not spare a damaged kidney, and, if administered, a sharp observation of the urine should be maintained. Personally, I should like to try the effects of repeated small transfusions of compatible blood, not merely for the cells it contains, but for its stimulating effect on the hemopoietic function. It goes without saying that foci of infection should be removed when determined.

RENAL LESIONS

Before essential hypertension was defined as an entity, the involvement of the kidney was called chronic Bright's disease; the elevated blood-pressure and traces of albumin with an occasional cast were deemed sufficient to justify that diagnosis; it was also called chronic interstitial nephritis, chronic diffuse nephritis, granular kidney and other names, which it was or was not as the autopsy disclosed. Two facts always confused the diagnosis of chronic nephritis in these cases: one was the astonishing variety of kidneys that were found, among which the normal kidney was most confusing, and the second was the long-continued and, in the vast majority of cases, the persistent absence of the symptomatology of chronic Bright's disease. Over and over again I have seen patients who long outlived the ablest diagnosticians of their day, who had granted them a term of life varying from a few months to two or three years.

The kidney status of these patients appeals to me in much this way. Very few die of uremia; in a certain number of advanced cases some degree of renal decompensation can be determined by laboratory methods and in these passive congestion plays a considerable rôle in the decompensation; the majority of patients have competent kidneys. They behave entirely differently from primary nephritis, true Bright's disease. What the kidney will show at autopsy is conjectural, as has been said, and another source of surprise is the lack of relationship between the kidney lesion and the symptoms.

I look upon these kidneys as the local expression of a widespread vascular change, arteriosclerosis, which, notoriously patchy, may hit the kidney early and hard in one case and not at all in another. The renal change is not inflammatory, but incident to slow nutritional changes secondary to insufficient blood supply through sclerotic vessels. Degeneration and connective tissue replacements and often marked shrinking occur; but the alteration is accomplished slowly; much parenchyma must be lost before evidences of renal incompetency occur. When the reserve tissue is largely sacrificed, circulatory inadequacy hastens the renal inadequacy.

In every case of essential hypertension it is advisable to determine the renal function; in every advanced or pronounced case it is imperative. The tests that I think sufficient are, first, the concentration test. Failure of concentration is one of the earliest falterings of renal function; this is particularly noticeable at night, resulting in a polyuria—nocturia.

The patient remains on his usual diet, or, failing these diuretic principles, there should be added coffee, oatmeal, some meat. He should drink no water between meals and none after the evening meal. He collects his urine at two-hour intervals; for example, he voids his urine at 8 A. M. and discards it; he voids at 10 A. M., 12 M., 2, 4, 6 and 8 P. M., collecting the entire amounts in separate bottles, each labeled with the hours of voiding. He collects the total of the night *after* 8 P. M. to and *including* that voided at 8 A. M. the next morning. This is *all* placed in one bottle, labeled 8 P. M. to 8 A. M. The physician measures and determines the specific gravity of each specimen. If concentration is unimpaired the night specimen should not exceed 400 to 450 c.c. under the conditions of the test and, because such night specimens are concentrated, the specific gravity is relatively high. It is the increasing quantity of their night urine that causes the frequency of micturition at night in renal cases. Day specimens vary greatly in quantity and specific gravity; normally this variation ranges through 8 to 10 points; if all specimens are persistently low, even preserving this range and accompanied by a large output, concentration is impaired. This is an extremely easy test, within the range of any practitioner, and gives early and significant evidences of renal inadequacy; but some impairment of concentration occurs in very many, of whom very few go on to a serious impairment of function.

In the second place, the Blood Chemistry should be done; that is, the concentration in the blood of secondary products of metabolism should be determined, the elimination of which it is the business of the kidney to accomplish, and an accumulation of which above certain levels impeaches the kidney. I determine *uric acid, urea nitrogen* and *creatinin.* I also determine *salt content* of the blood-plasma and the *sugar concentration* in the blood. The precise methods used in these analyses require a laboratory technic, and in communities remote from great centers, urge the establishment of laboratories that will serve physicians in a given radius.

Uric Acid.—The uric acid is often the earliest retained of the metabolites mentioned, but I have not found this relation to hold so constantly as many insist is the case; indeed, I doubt if it is the rule. Uric acid in a normal metabolism and normal kidney should not range above 3 mg. per 100 c.c. of blood; commonly it is under 2 mg. If the figures range very high while the urea nitrogen remains normal, one has to consider that faulty metabolism of the purin bodies (gout). It would be futile here to discuss the rôle of the metabolism and of the kidney in the uric acid accumulations of gout. One may say that moderately high blood uric acid in the absence of clinical gout, and on a diet containing usual quantities of purin, means early renal incompetency.

Urea Nitrogen.—Much more significant are high determinations of urea nitrogen or of non-protein nitrogen. Normal urea nitrogen ranges from 12 to 20 mg. per 100 c.c. (non-protein nitrogen 17 to 35 mg. per 100 c.c.). Above these figures accumulations of urea bespeak renal decompensation. Only rarely do I find this out of the range of the twenties and then apparently determined by passive congestion of the kidneys and coming down to normal when improvement of the circulation is established. Exceptionally I find it in the thirties and forties and due to the same cause; but higher figures than those almost invariably mean a perilous sacrifice of renal tissue and are commonly associated with uremia.

Creatinin.—Creatinin in normal blood should not exceed 2 mg. per 100 c.c. or 3 at the most. If one gets high creatinin and normal uric acid and urea nitrogen, the method is most certainly at fault, but, with an accumulating urea in the blood, high creatinin readings are ominous. It is the one constituent of the blood that has the highest prognostic significance. Very high creatinin accumulations are almost always, but not invariably, fatal.

Salt.—Salt accumulations have of late been magnified in importance in hyperpiesia. Allbutt and all others following him have laid great stress on the rôle of salt in at least maintaining or aggravating hypertension if not causing it. I was convinced years ago from observations made on my service at Bellevue Hospital that, in certain cases at least, the ingestion of considerable quantities of salt, 12 to 16 gm., could induce hypertensive crises, but I cannot follow Allen in the results he obtains by a salt-free diet. O'Hare and others have failed completely to affect the blood-pressure materially by salt-free diet. However, salt is certainly retained in a vast number of these cases, when nitrogenous constituents are unaltered. I take the normal salt content of the blood-plasma to be 562 mg. per 100 c.c. Figures above 600 mg. are exceedingly common.

Sugar.—Another curiously common accumulation in hyperpietics is sugar. It is true that the pancreatic arteries are involved in sclerotic processes (Allbutt), and it is true that diabetes does occur among hyperpietics, but the occurrence is not common; on the other hand, blood taken three to four hours after a meal will commonly show in hyperpietics 125 to 150 mg. of sugar per 100 c.c.

Phenolsulphonephthalein.—The third test that should be done is the phenolsulphonephthalein test. The method is simple and the convenient equipment for the test put out by many pharmaceutical houses makes it perfectly feasible for the general practitioner. Six-tenths mg. in 1 c.c. of water is injected into the subcutaneous tissue of, we will say, the thigh. There should be recovered in the next two hours from the urine of a normal kidney 60 to 80 per cent of the amount injected and most of that in the first hour. Delayed secretion (less the first hour than the second) or diminished secretion are both significant. Fifty to 60 per cent is debatable territory, but less so as it approaches 50. Under 50 per cent expresses incompetency.

It has been my experience that the lowering of phthalein output in cardiac decompensation is more marked than the accumulation of metabolites, and I feel little concern of impending uremia even in very low phthalein outputs if the urea nitrogen remains reasonably low in the blood. I have seen a 5 per cent phthalein output due to cardiac decompensation, as proved by subsequent events, in a man who rallied and lived for three years.

Acidosis.—In any advanced case the acidosis that may be present should be determined; among the various methods I prefer the CO_2 combining power of the plasma as determined by Van Slyke's method. Its normal should be not lower than 40.

I have said nothing of the Ambard coefficient and its modifications. They have not seemed to me to add anything to the combined urea nitrogen and phthalein methods.

Treatment.—What practical use shall we make of the information afforded? I have already discussed the diet established on the basis of economy, that is, relative rest, for any case of hyperpiesia. If there are abnormal quantities of any of the foodstuffs taken or their katabolic products heap up in the blood, it seems reasonable to restrict still more that particular food. If the uric acid is high, one should restrict the purins, until the results of such restrictions can be demonstrated by further blood analyses. This means the exclusion of all cellular organs, such as sweetbreads, liver, kidneys, brains, also beef, chicken and fowl as among the most offending; all meat, fish, oatmeal, peas, beans and lentils in marked retention.

If urea nitrogen is high, the patient should be put on a low protein diet, that is, a diet of cereals, breadstuffs, vegetables, fruit, cream, butter, oil; keeping the protein down to 35 gm. a day, unless the accumulation is high or uremia threatens, when, of course, still greater restrictions are necessary for the time being. How long to continue low protein and how much to increase it blood analysis affords information, but any patient who has once accumulated urea in the blood ought to remain on a low protein diet, 35 to 50 gm., or say ¾ to ⅘ gm. protein per kg.

When salt is high, the salt restrictions should be rigid until it is determined what effect such restrictions have upon the salt threshold; but we should beware how we persist in measures based on theory. Loss of appetite, loss of strength and increasing malaise should give the signal for a halt in any rigid dietary and let the patient's inclination argue that perhaps Dame Nature is not a bad consultant after all.

Patients with high blood sugar should have their carbohydrates restricted, particularly if they are overstout. In fact, the hypertension of the obese is often amazingly relieved by restrictions in carbohydrates, in other words, by loss of weight.

Water, too, has to be considered. Water the tissues must have, but what advantages there can be in drenching them, as is sometimes done, I fail to see. It has been demonstrated, to be sure, that water is an excellent diuretic and that large quantities ingested are followed by a flushing out of nitrogen for a short time; but this effect does not continue and certainly it would seem to put more work on the heart and kidneys to circulate and excrete it. I have always felt that thirst is the best guide for water intake, and that if thirst is not provoked by excessive salt and sugar intake its dictates may be heeded.

In all cases of accumulation of nitrogen I feel that the circulation is largely at fault; hence I believe that all cases that show high nitrogen in the blood should be put at rest in bed; put on digitalis and a restricted diet as defined above, with sedatives sufficient to secure sleep and relaxation.

When the kidney has passed the dead line, so to speak, we are faced with all the evils of true Bright's disease: a rapid deterioration, uremia in its varied manifestations, nausea, vomiting, diarrhea, headaches, delirium or stupor, twitching and convulsions.

Uremia

Of the nature of uremia we know nothing, for which reason there is no end of conjecture; but of its dependence on renal function we have felt firmly convinced. It is rather disconcerting then to have this solid foundation of our conception of uremia so rudely shaken as it seems to be in certain medical quarters; for the fact is that the symptom-complex agreed upon as uremia can occur in hyperpietics in whose kidneys at autopsy no pathological change can be demonstrated. Does this mean that uremia, although commonly *associated* with renal pathology, is not contingent upon it, or does it mean that identical clinical symptoms may be attributed to entirely different causes; that we have a pseudo-uremia, so to speak.

In my own experience I can say that I have seen classical "uremia" in fatal cases in which more than one detailed blood analysis, on different days after coma had obtained, showed normal figures for all constituents considered, showing thereby no evidence of renal decompensation; that I have seen the identical picture in another case, whose blood was laden with urea nitrogen, uric acid and creatinin, taken to mean profound renal distress; thus functional pathology shows the same discrepancies in the uremias of hyperpiesia as the structural pathology.

Delafield, one of the keenest students of kidney lesions this country has produced, a master clinician as well as pathologist, frankly abandoned an effort to attach clinical symptoms to definite kidney changes. It was for that reason that he divided chronic kidney disease into two great classes of chronic nephritis with exudation and chronic nephritis without exudation; the distinction was a clinical one of exudation (albuminuria, ascites, anasarca, etc.) and its absence. Of renal changes nothing more was incorporated in the nomenclature than that there were changes and with him many clinicians agreed. Most recently H. Batty Shaw in his studies states that he "is driven to the position of considering that *the actual physical state of the kidneys cannot be defined in a large proportion* *of cases by clinical examination!"* (The italics are his own.) He further shows among his cases that life may persist "until almost the last few glomeruli and tubules remain, death being brought about by the accompaniments of a complete anuria and what is known as uremia, or uremia may develop in individuals who have 96 per cent of normal glomeruli."

Batty Shaw also calls attention to the lack of relation between "albuminuric retinitis" and the extent of damage in renal tissue. He is arguing for what impresses us: that uremia is due to unknown toxins, which may and usually do damage the kidneys, but need not necessarily do so, but which affect particularly the brain tissue, by which they give rise to the symptom-complex of uremia. This widespread effect of the hypothetical toxins is seen in eclampsia; here, the toxin effect may be more marked on the liver than the kidney. Delafield in describing chronic nephritis with exudation (chronic parenchymatous nephritis) expressed his belief in a universal toxic effect on the vessels of which that in the kidney, with its consequent albuminuria, was but a local expression.

If this view prevails, it will help to explain the clinical uremias that we witness, associated with such discrepancies of renal pathology, structural and functional. There are a multiplicity of cerebral episodes ranging through a whole gamut from momentary dizziness, numbness of an extremity or aphasia, to convulsions and a full-blown clinical uremia, for the pathology of which there is no agreement among authorities. We hear of intermittent claudication from vascular spasm and, if the cerebral arteries have muscular coats, as they are said to have, they must subserve the usual function of muscle and spasm may well result; or intermittent claudication from kinking of branches of the cerebral arteries during hypertensive crises, and numerous brains have been demonstrated with thromboses of these branches making the explanation plausible. These minor manifestations have been attributed to small hemorrhages and the more carefully brains which have this history are sectioned and examined, the more certainly are multiple small hemorrhages, old or recent, determined. Coma, delirium, convulsions, paralyses occur in cases in which no lesion can be discovered. Some are attributed to areas of edema of the brain, others to effects of unknown toxins on specific centers. Some manifestations seem to be due to inadequate blood supply through sclerotic arteries with areas of softening.

Here again clinical signs cannot assure definite structural pathology in the brain any more than uremia can assure definite changes in the kidney.

How then can we differentiate between uremia and pseudo-uremia? Let it suffice in dealing with uremia that we accept a clinical picture and assume it due to an unknown toxin.

The acute manifestations of uremia are headache and sleeplessness; sudden blindness lasting hours or days and having no relation to albuminuric retinitis or other structural changes; seen in its most acute form in eclampsia; delirium and coma, coming on suddenly or gradually, with or without convulsions, temporary or continuing up to death; muscular twitchings and convulsions; vomiting, dyspnea, coming on in brief attacks or later continuing all day, but prone to occur in early morning hours; hemiplegias, monoplegias, aphasias, which may be transient or permanent; rise of temperature, usually low, but which may be hyperpyretic in exceptional cases. Usually there is a rise of pressure with the onset of the uremia. Delafield described a chronic form of uremia of gradual onset, not in repeated attacks, with headache, sleeplessness, delirium, stupor, coma, convulsions, a subnormal temperature, low blood-pressure, which once developing regularly continues until death.

Others have recognized these types of uremia as clinically distinctive and have believed them to be associated with a different cerebral pathology; it has been thought the chronic type of Delafield, the asthenic or what we might call the stuporous type, was due to edema of the brain, something like the "wet-brain" of chronic alcoholism; and that the acute type of Delafield or the convulsive type was accompanied by cerebral hyperemia and often by pin-point hemorrhages widespread throughout the brain substance. Foster believes that he has demonstrated a toxic substance in the blood of uremias of this type, which he could not recover from other types of uremia or from controls; he believes it to be an organic base forming crystalline salts with platinum and gold.

Treatment of Uremia.—As our knowledge of the cause of uremia and its pathology in the central nervous system is so scant, our treatment is largely empirical or at the most modified by more or less remote theoretical considerations. One of the theories not so remote is that there is a poisonous substance circulating in the blood; and that of the mechanisms for its disposal at the body's command there are the emunctories which should be utilized. It has been the long-established custom to have recourse to all those measures that increase these functions and the more vigorously or drastically, the more urgent the symptoms; hence catharsis, diuresis and diaphoresis, which will be dealt with presently.

Rest.—When patients begin to manifest those symptoms which are suggestive of uremia, headaches, restlessness, sleeplessness, disturbed gastric function, disturbed vision, and disturbed cerebration, they should be put to bed and as much rest and quiet maintained as possible. Careful consideration should be given to the state of the circulation and renal functional tests should be done. Retentions in the blood should be determined and the diet regulated somewhat in accordance. It should be very scant and simple under any circumstances, for example, milk, cereals, toast; but if there is decided nitrogen retention, the diet should for a time be fairly free from nitrogen. Water should be afforded in fair amounts, especially if the urine is scant, for when the kidney has lost its power of concentration to a considerable degree, it requires more water to eliminate a normal daily output of nitrogen. In patients whose tissues seem dry and whose stomach rejects fluid, the bowel may be used as a route by the Murphy drip method, the slow passage by catheter to the bowel of a reservoir of water from a suitable container or fountain syringe, or water may be given by a hypodermoclysis. In diarrheal cases, the fluid may be introduced into the subcutaneous tissues (hypodermoclysis) or into the vein as normal salt solution.

Sedatives.—It seems wise before beginning much therapy to allay in some measure the hyperexcitation of the central nervous system; for this we use, bromids, gr. xv to gr. xxx (1 to 2 gm.) 4.i.d. or, and more particularly if there is marked hypertension and much sleeplessness, chloral, gr. v. (0.33 gm.) q.6.h. to q.4.h. These may be used in combination.

Cathartics.—It has been the custom to use very drastic cathartics in these cases. If one had confidence that he were really eliminating appreciable quantities of toxins by this means, he might find justification for the great discomfort that these heroic measures entail. I am not so convinced. I am content to keep the bowel thoroughly cleansed by a preliminary dosage with Epsom salts or compound jalap powder and by daily irrigations or enemata. I must concede that vigorous purging is traditional and still advocated by able clinicians.

Diaphoresis.—Many clinicians continue the time-honored sweatings in uremia. This is effected by the hot pack given two or more times a day, and continued from fifteen minutes up to an hour. Certain it is that some patients feel much relieved by the procedure, have less headache, sleep better and enjoy them. For such I order them, but one can explain the benefits derived from the diaphoresis without appealing to the elimination of toxins. However, if the patient is rendered more uncomfortable, as many are, I think there is not sufficient justification for continuance of the measure on theoretical grounds alone. Some patients may collapse in the pack; and therefore all should be closely watched. Diaphoresis may be induced by pilocarpin given hypodermically in doses of gr. 1/10 (0.006 gm.). When it operates, the result is often extraordinary—water floods out of the pores. It is not altogether free from danger and some observers declare that edema of the lungs is too often induced by it to justify its usage. Pilocarpin in lesser doses has been used in conjunction with the pack.

Diuresis.—If toxins circulating in the blood are eliminated the kidney is probably the emunctory called upon, the skin and bowel at the most acting vicariously. Diuresis may be effected by improving an impaired circulation, or by stimulating the secretory apparatus in the kidney itself. The great value of digitalis as a diuretic depends on its effect on the circulation, and as the circulation is so commonly impaired (we would better say usually impaired in these cases), it is indicated as a diuretic in uremia. It is to be used as described in Cardiac Decompensation. The preparation is indifferent, provided the dosage is sufficient. The reputation of the infusion as a diuretic rested on the fact that the textbook dosage of the infusion of digitalis was much larger than that of any other preparation, and, in fact, alone approached an adequate dosage.

Of the second class of diuretics, the purin derivatives are alone worthy of consideration. They are, however, not to be carelessly administered as there is evidence that they are irritant to the renal epithelium and may do damage. This danger is greater in acute nephritis and in acute exacerbations of chronic nephritis than in the chronic stages. There are three that may be considered: caffein, theobromin and theocin. Of these I think the last is the most potent and also the most irritating. The dose is gr. iss to v (0.1 to 0.33 gm.) three times during the day and one day only. If diuresis does not follow this dose, more entails danger.

Diuretin, the double salt of theobromin sodium and sodium salicylate is safer. It may be given in doses of gr. v to gr. viiss (0.33 to 0.5 gm.) three times a day. It is not wise to continue the dose longer than two days in succession. Caffein in doses of gr. i to gr. iiss (0.06 to 0.15 gm.), or gr. v (0.33 gm.) of the double salts of sodium benzoate or salicylate may be used for one or two days three times during the day. One objection is that it is likely to make the patient wakeful. One must not expect an abundant diuresis if no collections of fluids are demonstrable either in the subcutaneous tissues or in the serous cavities; how much there will be depends on the water ingested and *water,* indeed, is the best and safest diuretic, and the rationale of its administration has been touched on above.

Of the alkaline salts as diuretics, I have little to say. When salt is retained the salts with sodium bases should be withheld, potassium being preferred. I should advocate them only when I could demonstrate an acidosis, or had reason to suspect one.

Venesection.—I feel convinced that this is a most valuable measure in the cases with which we are dealing; one may withdraw 400 to 800 c.c. (13 to 26 oz.) and in exceptional cases more. I know of no way in which a toxin, if present in the blood, can be so surely eliminated. After such bleeding from the plethoric or, those with a high red cell count, fluid may be replaced by mouth or bowel, or if this is not feasible on account of vomiting and diarrhea, by hypodermoclysis or injection into the vein of a like amount of normal saline solution. If the patient is anemic it is better to replace the blood withdrawn by healthy, compatible blood by transfusion.

Transfusion.—This has always appealed to me strongly on theoretical grounds. It replaces a fluid better than any artificial fluid that can be devised for salt balance and viscosity, and contains unpoisoned red cells, normal foodstuffs and normal antibodies to combat the ever threatening secondary infections. Washing and replacing the patient's own red blood-cells offers too many technical difficulties and no advantages that I can discern. Perhaps some day the experiment in animals by which an artificial kidney is intercalated into the blood-stream and by which nitrogenous substances and presumably toxins are dialyzed off may be applicable to the human; who knows? Some have advocated bleeding these patients white and replacing by healthy fluid—it sounds a bit heroic.

Lumbar Puncture.—With twitchings or convulsions I advise a spinal puncture. The fluid is usually under decided pressure, contains the same nitrogenous bodies as the blood and in almost, though not quite, the same concentration; it in all probability contains the unknown toxic substance in much the same concentration. It does seem reasonable to remove it from direct contact with the nerve tissue on which it exerts such a deleterious effect. At any rate the results are often fortuitous.

Treatment of the Convulsions.—The treatment of convulsions is the treatment of the uremia itself as set down above, including venesection and lumbar puncture. In addition drugs are indicated. With the twitching morphin hypodermically is indicated in doses of gr. ¼ (0.015 gm.) to gr. ⅓ (0.020 gm.). If the convulsion is on, the best measure is the administration of chloroform (some prefer ether) until the seizure is quelled and this backed up by morphin, as above, to prevent or delay its recurrence; a folded napkin or similar soft substance should be placed between the teeth to prevent biting the tongue.

Convulsions, epileptiform in character, may occur in the hyperpietic from time to time and dissociated from other uremic manifestations, but diagnosed as uremia. In one of my patients they occurred only at night. Loss of consciousness and coma for hours, thought to be uremic, occur in others, but the patient promptly recovers and may never have another. Such attacks may show no accompanying paralysis or paresis or other uremic manifestations. They certainly seem of vascular origin; may they not be due to cortical irritation from old vascular accident?

Headaches are commonly uremic, though not necessarily so; when they are they may be relieved by a brisk cathartic or a sweat; such drugs as phenacetin or aspirin may afford some relief and even the hypnotics, such as trional and veronal, have been credited with ameliorating them; morphin may be required in the more severe cases. The restlessness and sleeplessness of uremia had best be met with morphin in doses of gr. ⅛ to gr. ¼ (0.008 to 0.015 gm.).

Suppression of Urine.—This occurs in a certain few of the cases. It usually means a kidney far advanced in structural change, or it may be precipitated by a renal calculus. The stock procedures are to apply heat to the lumbar region—wet or dry—and some cup the parts.

Diet.—Fluid should be restricted but not cut out. A Karell diet of 800 c.c. of milk in 200 c.c. portions will be suitable for an adult. In addition one may have recourse to a vigorous catharsis with Epsom salts or compound jalap powder, to sweating (diaphoresis) by methods mentioned above, and to venesection.

TERMINAL INFECTION

The rapid breakdown, one might justifiably call it dissolution, in many of these cases has been dwelt upon. Batty Shaw would emphasize the rôle played by infection in many terminal cases and I am inclined to think that we too often overlook or slur the evidences of such.[1]

If pleurisy and pericarditis may be taken to express infection, they are far from unusual. I have been struck by the extensiveness of these serous membrane involvements and their persistence when pain or when other evidence of the inflammation is absent and only outspoken physical signs tell the tale.

Temperature may be present or absent, the pulse may be accelerated and the previously high blood-pressure may fall to normal or near it. In fact, such a fall should lead to the strong suspicion of infection. The lung is prone to attack; we find bronchitis, bronchopneumonia, pulmonary infarct, dry pleurisy, pleurisy with effusion or empyema; pericarditis in different degrees of severity, even hemorrhagic septicemia, meningitis, otitis, petechiæ, hematuria, retinal hemorrhages.

Some of these cases would seem to be the *result* of the complete breakdown of the mechanism of defense, as we see it in the late stages of many chronic diseases; others would seem to occur early enough to play a rôle in shortening a life that might otherwise be prolonged; *for this reason all foci of infection should receive attention in the hyperpietic to prevent such disasters;* teeth, tonsils, ears, sinuses, gastro-intestinal tract, respiratory tract, genito-urinary tract, hemorrhoids, fistulæ should be investigated.

Such foci of infection besides giving rise to the above expressions of infection are a menace to the heart; so, too, are all fresh infections, for the heart may be directly attacked or undergo a depreciation through the action of toxins or from loss of tone in the peripheral vessels, through toxins, "so that it beats the air in vain" (Allbutt). Allbutt has also called attention to the liability of a heart, even previously good, to suffer strain after effort during convalesence from acute infections.

---

[1] The importance of infection in initiating serious complications in hypertensives, cardiacs, nephritics and diabetics has been largely overlooked by the profession in general.—Editor.

CONCLUSION

Hyperpiesia is a state, whose earliest expression is an increased blood-pressure, due to an unknown toxic agent; a state in which the brunt of the disorder falls on the heart and blood-vessels, leading to impairment of these structures and certain important organs they subserve. The symptomatology is referable to impairment of heart, vessels and organs concerned, and the treatment physical, mental, cardiac, vascular, renal and metabolic rest; all other measures are mere succedanea.

SUMMARY OF TREATMENT

I. Cases Showing no Symptomatology

1. Early cases (said to be curable—Allbutt)
    a. Rest in bed.
    b. Diet (see below).
    c. Sedatives (see below).
        (1) Mental therapy.
        (2) Elimination of worry.
        (3) Drugs.
    d. Gradual return to normal activities under observation.
2. Cases occurring in the first three decades and any case for purposes of observation, observing restrictions noted on page 382. May be treated as early cases.
3. Later cases
    a. Avoid pernicious interference and the establishment of introspection in the patient by too frequent observation.
    b. Determine cardiac and renal efficiency.
    Instruct the patient in the significance of his disorder.
    Afford reassurance and reëxaminations at intervals of three to six months.
    c. Avoid additional obligations.
    d. Plead for more frequent holidays and diversions.
    e. Diet. Avoidance of excess as shown by overweight.

II. Cases Showing a Symptomatology

1. *Habits of Life, Rest*
    Only exceptionally necessary to interrupt business activities.
    Avoid taking over new enterprises.
    Shorten the number of hours at his business.
    Cultivate the habit of week-ends.
    Take more frequent short vacations and one long vacation at least once a year.
    Lie down in the middle of the day for a few minutes' sleep or relaxation.
    Rest awhile after the evening meal.
    Spend more hours in bed each night.
2. *Clothes and Temperature*
    Keep warm, but do not overload.
    Light weight woolen or silk next to the skin.

Avoid exposure to the sharp cold without adequate overclothing.
Keep even temperature in office and home.
Avoid crowded conveyances.
Fresh air for the sleeping room, but avoid exposure during the night by wearing woolen night clothes.

3. *Baths*
Theoretically warm bath (not hot) is best and best taken at night.
A lifetime habit of cold bathing, if the reaction is good, may be continued.

4. *Exercise*
Previous habits must be considered.
Reaction to exercise must be studied.
For men of sedentary habits walking or not too strenuous golf or horseback-riding.
More active forms of exercise and sport for those who are accustomed to them.
Avoid excessive and sudden effort.
Avoid mental excitement.

5. *Diet*
Quantity most important.
If overweight, restrict foods to induce a gradual loss until normal weight is approximated.
Avoid excessive restrictions in those who lose weight slowly, in those in whom weakness develops or an impairment of digestion.

6. *Proteins*
May be diminished below the ordinary intake with benefit.
One modest helping once a day of meat, fowl or fish.
Rule—0.8 gm. protein per kg. of body-weight, allowance being made in the extremely obese. (Foster)
Some patients are more comfortable for excluding red meats.
When renal involvement occurs and nitrogen is retained, nitrogen is to be sharply restricted in the diet until normal figures are attained in the blood.

7. *Carbohydrates*
Should be so restricted as to maintain normal weight.
If blood sugar is high should be restricted.
Occasionally involvement of the pancreas induces a dietetic consideration.

8. *Fats.*
Restricted only in case of overweight.

9. *Salt*
Found retained in the blood of a large percentage of cases.
Salt commonly ingested far beyond physiological needs.
Under all circumstances eliminate salt at table, but allow enough in cooking to make food palatable.
If salt is retained in the blood, unsalted butter should be used, no salt meat or fish allowed or soups with meat stock.
Much more rigid restrictions, that is, a salt-free diet is established by some who believe that salt plays a considerable rôle in maintaining pressure.

10. *Fluids*
Limit fluids to 1½ to 2 quarts a day, but after all be guided by thirst.
See also summary on text-page.

11. *Heart*

    Palpitation or discomfort from overaction.
    - a. Mild sedatives, for example, bromids, gr. xv (1 gm.) three or four times a day for three or four days.
    - b. Luminal gr. ss (0.03 gm.) two or three times a day until results are obtained.

12. *Cardiac Inefficiency*
    - a. Rest. Complete or partial depending upon seriousness of symptoms.
    - b. Restriction of diet.
    - c. Attention to the bowels.
    - d. Mild sedatives in the excitable.
    - e. Hypnotics for insomnia (see above).
    - f. Venesection in the plethoric in hypertensive crises 12 to 16 ounces.—350-500 c.c.
    - g. Digitalis—moderate dose, for example, gr. vi (0.4 gm.) for four or five days. Then gr. iss-iii (0.1 to 0.2 gm.) as long as needed.
    - h. With improvement—a holiday and new environment. Work resumed under observation.

13. *Insomnia*
    - a. Trional gr. v (0.33 gm.) to be repeated once or twice.
    - b. Adalin gr. v (0.33 gm.).
    - c. Chloral gr. xv (1 gm.).
    - d. Medinal gr. v (0.33 gm.).
    - e. Veronal gr. v (0.33 gm.).

14. *Dyspnea with Acute Onset*
    - a. Cardiac asthma.
        - (1) Morphin gr. ⅛ to ¼ (0.008 to 0.016 gm.).
        - (2) Rest in bed.
        - (3) Restricted diet.
        - (4) Digitalis.
    - b. Dyspnea of acidosis.
        - (1) Bicarbonate of soda, teaspoonful doses, 5 grams at 2 hour intervals until urine is alkaline. Two doses should alkalinize a normal urine.
    - c. Uremic dyspnea (see uremia).
    - d. Paroxysmal dyspnea.
        - (1) Nitroglycerin gr. 1/50 to 1/10 (0.0012 to 0.006 gm.) under the tongue best or under skin.
        - (2) Morphin hypodermically gr. 1/6 to 1/3 (0.01 to 0.02 gm.).

15. *Pulmonary Edema*
    - a. Nitroglycerin (same as above).
    - b. Venesection of about a pint of blood from plethoric individuals.
    - c. *Strophanthin if patient has not had digitalis,* ½ mg. (gr. 1/120) in muscle or into vein.
    - d. Digitoxin 1½ mg. (gr. 1/40) into vein (see text).
    - e. Digitalis gr. v to vii into vein (0.33 to 0.5 gm.).
    - f. Atropin sulphate gr. 1/50 to 1/33 (0.0012 to 0.0018 gm.) into muscle or vein.
    - g. Cupping (see text).
    - h. Morphin sulphate gr. ⅛ to ¼ (0.008 to 0.016 gm.).

16. *Pulmonary Infarct*
    - a. Morphin gr. ⅛ to ¼ (0.008 to 0.016) (see text).

17. *Angina Pectoris—Cardiac Pain*
   a. Rest in bed, or couch or easy chair for two, three or more weeks or until attacks, that come on even at rest, disappear.
   b. Mild sedatives—bromids gr. xv (1 gm.) three or four times a day for a few days. Then replace gradually by luminal gr. ss (0.033 gm.).
   c. Mental rest. Avoidance of excitement, worry, business details, etc.
   d. Insomnia. Bromids and luminal. Trional gr. v, x or xv (0.33 to 0.6 to 1 gm.) or 2 gr. every two hours during day. Chloralamid gr. xxx (2 gm.). Do not use a hot menstruum. Adalin gr. v (0.33 gm.). Medinal or veronal gr. v to viiss (0.33 to 0.5 gm.). In rebellious cases chloral hydrate gr. x to xv (0.66 to 0.1 gm.).
   When pain is present, morphin gr. ⅛ to ¼ (0.008 to 0.016 gm.). Avoid latter in nagging type in women.
   e. Diet—light.
   f. Bowels—mild cathartics or enema.
   g. Digitalis (see text).
   h. After rest period of three to six weeks allow patient about under observation. First on a level, then on incline (see text).

18. *Treatment of Acute Type of Angina*
   Patient should be instructed how to treat himself in emergency.
   a. Nitrites.
      (1) Amyl nitrite "pearls" M. iii to v crushed in handkerchief, napkin or towel and inhaled. Effect—immediate.
   b. Nitroglycerin gr. 1/100 to 1/25 as spirits of glonoin in tablet form under the tongue to be absorbed there. Almost as prompt as amyl nitrite.
   c. Sodium nitrite gr. i to ii (0.06 to 0.12 gm.) every two or three hours.
   a. Erythrol tetranitrate gr. ¼ to gr. i (0.015 to 0.06 gm.) every two or four hours. The last two when the attacks are nagging and frequent.
   e. Morphin. When the nitrites fail, gr. ¼ to ⅓ (0.015 to 0.02 gm.). Repeat one-half the dose in fifteen minutes if necessary.
   f. Atropin. Combine with morphin in doses of gr. 1/100 to 1/150 (0.0006 to 0.00045 gm.). Lessens the danger of cardiac inhibition through the vagus.
   g. Chloroform. If nitrites fail and morphin not at hand, inhaled. Best self-administered. Use wide-mouth bottle containing moistened sponge. Bottle drops from hand as patient becomes drowsy.
   h. Chloral. May relieve long attacks or prevent recurrence (Mackenzie), gr. x to xv (0.66 to 1 gm.) at night or gr. v (0.33 gm.) four times a day.
   i. Oxygen. Inhaled. Said to afford some relief.

19. *Lesser Attacks—Angina Minor*
   a. Hot drinks.
   b. Water.
   c. Soda bicarb. ʒi (4 gm.) to glass of water.
   d. Simple carminatives such as spirits of peppermint five or six drops with bicarbonate.
   e. A little whisky or brandy in hot water.
   f. Mustard plaster or leaf or hot fomentations to the precordium.

20. *Circulatory Collapse*
   a. Strophanthin ¼ mg. (gr. 1/240) into muscle or vein.
   b. Digitalis gr. iii to v (0.2 to 0.33 gm.) into the vein or into the muscle.
   c. Camphor in oil gr. v (0.33 gm.) into muscle—less value.

d. Caffein, double salt of caffein and sodium benzoate or salicylate gr. v to viiss (0.33 to 0.5 gm.) less valuable than digitalis bodies.
e. Aromatic spirits of ammonia ʒi (4 c.c.) in water by mouth.
f. Stronger water of ammonia cautiously inhaled.

21. *General Treatment of Angina*

The same as that laid down for essential hypertensions with added precaution.

a. Avoid excitement.
b. Avoid worry.
c. Avoid hurrying.
d. Avoid excessive physical or mental effort.
e. Avoid taking inclines.
f. Avoid walking in face of wind, especially cold wind.
g. Avoid cold air and chilling of skin.
h. Insist on warm outer garments even on brief exposure to cold air.
i. When allowed to work, insist on shorter hours, more rest and more vacations.
j. Insomnia.
    (1) Treat with bromids, luminal, trional, adalin in the smallest doses effectual (see text).
k. Diet that of hyperpietics.
    (1) Suspect carbohydrates if there is much flatulency.
    (2) Alcohol, tea, coffee, and tobacco are better eliminated or the effects carefully studied.
    (3) Avoid large meals, especially at the end of the day.
l. Infection.
    (1) Search for foci.
    (2) Eliminate where possible.
    (3) Use vaccine in suitable cases.
m. Gastro-intestinal disturbances (see text).
    (1) Relief should be prompt.
    (2) Carminatives to expel the gas, that is, spirits of peppermint with or without bicarbonate of soda, 6 drops of peppermint, 1 dr. of soda (4 gm.)—preferably hot water.
    (3) Hoffman's anodyne.
    (4) Spirits of chloroform.
    (5) Compound spirits of lavender.
    (6) Aromatic spirits of ammonia in ʒi (4 gm.) doses or in combination, in water.
    (7) Bromids gr. xv to xxx (1 to 2 gm.) as sedatives.
n. Constipation.
    (1) Regulate food.
    (2) Furnish roughage.
    (3) Use mineral oil.
    (4) Agar.
    (5) Bran.
    (6) Figs.
    (7) Prunes.
    (8) Grapes.
    (9) Dates. All helpful.
    (10) Use mild drugs—cascara, aloin, compound licorice powder.
    (11) Avoid straining at stool.

    (12) Exercise. Walking on a level, later slight inclines.
    (13) Never disregard discomfort.
  o. Drugs for continued use.
    For decompensation small doses of digitalis grs. iss-iii (0.1 to 0.2 gm.) a day. Watch for any increasing pain under digitalis.
  p. Atropin.
    To prevent inhibitive shock gr. 1/200 to 1/100 (0.0003 to 0.0006 gm.) every four hours.
  q. Supprarenal extract.
    (1) For nagging pains (see text).
    (2) Arsenic.
    (3) Inhalation of oxygen.
    (4) High frequency current. Are all advised (see text).

22. *Coronary Thrombosis and Embolism*
  a. Rest.
  b. Morphin gr. ¼ hypodermically (0.016 gm.).

23. *Decompensation*
  Met by strophanthin ½ mg. (gr. 1/120), of the crystalline product into vein or muscle followed by digitalis gr. vi to x a day (0.4 to 0.66 gm.) for three days.

24. *High Blood-Pressure Stasis*
  a. Rest.
  b. Restrict food and fluid.
  c. Venesection, especially if there be a hypertensive crisis, 300 to 900 c.c. (10 to 30 ounces) (see text).
  d. Digitalis.
    The text should be carefully read on this subject (see page 406)

25. *Auricular Fibrillation*
  a. Rest.
  b. Morphin for sleep hypodermically gr. ¼ (0.015 gm.).
  c. Diet. Strict Karell of 800 c.c. (28 ounces) for the first three or four days followed by a dry diet.
  d. Digitalis. Consult text carefully.

26. *Diuresis*
  a. Theocin gr. v (0.33 gm.) three times a day for three doses.
  b. Diuretin gr. viiss (0.5 gm.) three times a day for two or three days.

27. *Cardiac Failure with Edema*
  a. Rest in the most comfortable position.
  b. For insomnia.
    (1) Bromids.
    (2) Adalin.
    (3) Trional.
    (4) Chloralamid in above doses.
    (5) In the urgent cases, morphin sulphate hypodermically gr. ¼ (0.015 gm.). Repeat with one-half the dose if necessary.
  c. Diet.
    (1) Karell the best; 800 c.c. of milk in 200 c.c. (7 to 8 ounces) quantities.
    (2) Add no more fluid than relieves an urgent thirst.
    (3) If other food is given the diet should be dry and salt-free.

- d. Digitalis.
  (See Auricular Fibrillation; also text.)

28. *Tardy Diuresis*
    (See above and see text.)
29. *Edema*
    (Southey tubes, scarification, and see text.)
30. *Hydrothorax*
    Thoracentesis (see text).
31. *Hydropericardium*
    (See text.)
32. *Ascites*
    (See text.)
33. *Paroxysmal Tachycardia*
    - a. Pressure on the vagus.
    - b. Ocular pressure.
    - c. Simple maneuvers stimulating the vagus (see text).
    - d. Drugs. Digitalis group (see text).
    - e. Diet. Simple and designed to lessen flatulency.
34. *Premature Systoles*
    - a. Rest.
    - b. Sedatives.
      - (1) Bromids.
      - (2) Luminal gr. ss (0.03 gm.) t.i.d.
    - c. Elimination of tobacco, tea, coffee and alcohol.
    - d. Nitrites have been advised by Mackenzie.
35. *Vascular Accidents*
    (See Angina Pectoris, Coronary Thrombosis or Infarct)
36. *Cerebral Accidents*—premonitory symptoms
    - a. Rest.
    - b. Avoidance of business and sources of worry.
37. *Transient Paralysis*
    - a. Rest in bed.
    - b. Saline cathartics.
    - c. Restrict diet.
    - d. Restrict *fluid.*
    - e. Sedatives.
      Bromids gr. xv (i gm.) three or four times a day.
    - f. Restlessness and insomnia.
      Morphin sulphate hypodermically gr. 1/8 to 1/4 (0.008 to 0.015 gm.).
    - g. Restlessness and insomnia with increased hypertension.
      - (1) Chloralhydrate gr. v (0.33 gm.) with or without bromids every four to six hours.
      - (2) Nitroglycerin gr. 1/100 to 1/50 (0.0006 to 0.0012 gm.).
      - (3) Erythrol tetranitrate gr. 1/2 (0.03 gm.) every three hours.
      - (4) Sodium nitrite gr. 2 to 3 (0.12 to 0.2 gm.) every four hours.
      - (5) Phlebotomy—12 to 16 ounces, 350 to 500 c.c.
38. *Severe Apoplexies*
    - a. Rest.
    - b. No food in the early hours or days of unconsciousness.
    - c. Water by mouth if swallowed; if not, by bowel, for example, a Murphy drip or small injections.

- d. Care of mouth, teeth, tongue, pharynx, nose with mild antiseptics on swabs or sprays.
- e. Bowels. Enemata or irrigations.
- f. Restlessness. Morphin sulphate hypodermically.
- g. Excessive hyperpiesia. Nitroglycerin or other nitrites (see above).
- h. Phlebotomy in the plethoric cases.
- i. Convulsions.
  - (1) Morphin sulphate gr. ¼ (0.008 gm.). Repeat with one-half the dose shortly if necessary.
  - (2) Lumbar puncture. Should be done slowly until pressure of fluid is materially decreased.
- j. Later feeding. Non-stimulating food at first in small quantities (see text).
- k. Prevent contraction of muscles.

39. *Mesenteric Thrombosis*
    - a. Acute case—surgery.
    - b. Intermittent claudication.
      - (1) Morphin hypodermically.
      - (2) Nitrites—less helpful.
      - (3) Hot cloths to abdomen between the attacks.
      - (4) Avoid drastic cathartics, warm enemata—not hot.
40. *Thrombosis of Peripheral vessels*—chiefly in the extremities
    - a. Elevate the part.
    - b. Keep warm.
    - c. If gangrene appears, hot, dry air.
    - d. After demarcation, amputation or excision.
41. *Intermittent Claudication*
    - a. Warmth.
    - b. Gentle massage.
42. *Hemorrhages*—of the brain (see text)
43. *Epistaxis*
    - a. Moderate hemorrhage, let alone.
    - b. Severe hemorrhage plug the nostrils (see text).
    - c. Hemostatic tissue extracts applied on tampon.
    - d. For restlessness and alarm. Morphin hypodermically.
44. *Hemorrhages*—rarely require interference
45. *Anemia* (see text)
46. *Nephritis*
    - a. Determine renal functions.
    - b. If uric acid is retained, restrict the purins (see text).
    - c. If nitrogen is retained, restrict the proteins.
    - d. When salt content of the blood is increased, restrict the salt.
    - e. When blood sugar is increased, and especially in the overstout, restrict carbohydrates.
    - f. If loss of appetite and strength ensue disregard theory and arrange dietary more to the patient's liking.
    - g. Restrict fluids.
47. *Renal Decompensation*
    - a. Rest in bed.
    - b. Diet restricted as above.
    - c. Sedatives for sleep and relaxation.
    - d. Digitalis.

48. *Uremia*
  - a. Rest and quiet vigorously maintained.
  - b. Low nitrogen diet and if much retention, diet fairly free from nitrogen.
  - c. Water in moderate amounts.
  - d. In patients with dry tissues and who are vomiting use the bowel by the Murphy drip or hypodermoclysis.
  - e. In diarrheal cases hypodermoclysis or infusions of normal salt solution.
  - f. Sedatives.
    Bromids gr. xv to xxx (1 to 2 gm.) four times a day.
  - g. Marked restlessness and sleeplessness.
    Chloral gr. v (0.33 gm.) every six or every four hours. Chloral and bromids may be combined.
  - h. Cathartics. For author's views see text.
  - i. Diaphoresis. For author's views see text.
  - j. Hot packs. Pilocarpin.
  - k. Diuresis.
    - (1) Digitalis (see cardiac decompensation and see text).
    - (2) Theocin gr. iss to v (0.1 to 0.33 gm.) three times one day.
    - (3) Diuretin gr. v to viiss (0.33 to 0.5 gm.) three times a day. Not wise to continue longer than two days.
    - (4) Caffein gr. v of the double salts (0.33 gm.) of sodium benzoate or salicylates three times a day for two or three days.
  - l. Venesection.
    - (1) 400 to 800 c.c. (13 to 26 ounces).
    - (2) Fluid replaced by mouth or bowel or hypodermocylsis or normal saline into vein.
    - (3) If patient is anemic replace blood withdrawn by transfusion.
    - (4) Lumbar puncture with twitching and convulsions.
  - m. Convulsions.
    - (1) Venesection.
    - (2) Lumbar puncture.
    - (3) Morphin sulphate hypodermically gr. ¼ to ⅓ (0.015 to 0.20 gm.).
    - (4) During convulsions—chloroform (some prefer ether) until seizure ceases. Follow by morphin as above.
  - n. Headaches.
    - (1) Brisk cathartic.
    - (2) Hot pack.
    - (3) Phenacetin.
    - (4) Aspirin.
    - (5) Some have advised trional and veronal.
    - (6) In more severe types morphin.
  - o. Restlessness and sleeplessness.
    Morphin gr. ⅛ to ¼ (0.008 to 0.015 gm.).
  - p. Suppression of urine.
    - (1) Heat to lumbar region.
    - (2) Cups—wet or dry.
    - (3) Diet—
      Fluid restricted, but not cut out.
      Karell diet—800 c.c. of milk.
      Vigorous cathartics with Epsom salts or compound jalap powder.
  - q. Diaphoresis.
  - r. Venesection.

CHAPTER XVIII

TREATMENT OF SYPHILITIC AORTITIS AND AORTIC ANEURYSM

Thomas McCrae

SYPHILITIC AORTITIS

The Treponema pallidum frequently settles in the aorta with variable resulting changes. In some cases there is an acute aortitis, in others a chronic aortitis (dilatation of the aorta) which results from a previous acute process or comes on gradually without any acute features. The process affects particularly the middle coat of the aorta with small round cell infiltration, especially around the vasa vasorum. It may be diffuse or most evident in slightly raised areas with a yellow tinge. The first part of the arch is especially likely to be affected and this often means involvement of the aortic valves and the orifices of the coronary arteries. Syphilitic aortitis occurs with acute features most often in young adults and not infrequently within a short interval after the primary infection.

Clinical Features.—Pain is the most common symptom and is usually referred to the upper part of the sternum. It may radiate to either arm but rarely has the severity of angina pectoris. Not infrequently it occurs in paroxysms sometimes brought on by effort. There may be a sense of thoracic oppression and dyspnea. If the enlargement of the aorta is marked symptoms due to pressure, such as cough, may be present. As disease of the aortic orifice and myocarditis frequently occur with it, the symptoms of these may predominate. The marked pulsation in the neck is a striking sign of the condition, especially in the suprasternal notch where the aorta may be seen and felt. There may be visible pulsation in one or all of the upper two interspaces on both sides. There is dulness over the manubrium and to the right or left or both sides of the sternum in the first two interspaces. The second aortic sound, if present, may have a musical bell-like quality. The X-ray examination gives a very characteristic picture.

Treatment.—It is important to recognize that treatment evidently has two objects: (1) to kill the spirochetes, and (2) to limit the damage done to the aorta and restore it as far as possible to its previous condition.

As regards the destruction of all spirochetes we can say very little—we hope it may be possible. The complete restoration of the aorta is impossible but if the damage is not too great and further progress is stopped, much is accomplished. Early recognition and proper treatment should reduce the number of aneurysms. In general, treatment is palliative but not completely curative.

Acute Syphilitic Aortitis.—Early recognition is important if anything useful is to be done. The condition occurs especially in young adults and often within a year or two of the primary infection. Pain is a prominent symptom and the signs are usually clear. Active treatment is important as the aortic valves and the orifices of the coronary arteries are usually involved in the process, and first place should be given to mercury which should be pushed vigorously. The best method is by inunction, beginning with 2 gm. (gr. xxx) of mercurial ointment daily and doubling the amount in a few days. It is wise to give a course of thirty inunctions. At the same time potasssium iodid should be given persistently, beginning with 2 gm. (gr. xxx) a day. This may be increased, but massive doses are usually not advisable and a maximum dose of 4 gm. (gr. lx) a day is generally sufficient. This should be kept up for a month, after which the dose can be reduced, but it is wise to give 1 gm. (gr. xv) daily, with short intermissions of a few days, indefinitely.

The question as to the wisdom of giving some arsphenamin preparation has to be decided for each patient. It should always be given cautiously and signs of severe myocardial disease usually contra-indicate its use, which has some danger of harm. A dose of 0.2 gm. of neo-arsphenamin is safe, as a rule, and if it does not cause any disturbance, should be repeated every five to seven days for four doses. This should be followed by another course of mercury as before and after this the course of neo-arsphenamin is repeated. The decision as to subsequent administration of mercury and arsenic should be decided by the general rules of treatment in syphilis. The general condition of the patient should be considered and the treatment modified if necessary. Disturbance from mercury or arsenic should lead to the reduction of the dosage of the former and the stopping of the latter.

General Measures.—The patient should be at absolute rest in bed in the position in which he is most comfortable. The diet should be of liquids and soft foods at first, with the amount of fluids not over 1,500 c.c. in the twenty-four hours. The bowels are to be kept freely open, for which cascara, senna, mineral oil and salines can be used as proves best.

Symptomatic Treatment.—The pain is sometimes severe and sedatives are often required. Codein may be given by mouth in doses of 0.016 gm. to 0.06 gm. (gr. 1/4 to gr. i) or morphin sulphate 0.016 gm. (gr. 1/4) hypodermically if the codein is not sufficient. Heat or counterirritation over the upper sternal region is sometimes useful. If there are signs of pressure, as in aneurysm, venesection is helpful, from 300 to 500 c.c. being withdrawn. The iodid usually has a marked influence on the pain.

Chronic Aortitis.—This is not always syphilitic but if proved or strongly suspected of being so, the treatment should be practically the same as in the acute form.

AORTIC ANEURYSM

The problem of aortic aneurysm is almost always that of syphilitic infection which has resulted in serious damage to the vessel. The clinical features are influenced by the situation and size of the aneurysm for they are due largely to the effects of pressure. The most important symptom is *pain* which is rarely absent. Its character and occurrence vary but it is often severe, especially if the vertebræ are being eroded. Anginal attacks may occur. Sometimes the pain is persistent and severe. Dyspnea and cough are frequent, due usually to pressure on the air passages; the cough may be paroxysmal and if due to or associated with recurrent laryngeal nerve paralysis has the so-called goose or brassy quality. Difficulty in swallowing is due to pressure on the esophagus. Hemorrhage may occur externally when the aneurysm "points." Occasionally there is bleeding into the air passages over considerable periods of time.

The physical signs may be classified as: (1) those made out by the ordinary methods of examination, and (2) pressure signs.

On *inspection* there may be a visible impulse in one or more interspaces or a diffuse visible heave, especially of the upper sternum. In some cases there is a prominence due to the aneurysm eroding the chest wall. On *palpation* an impulse, a shock, usually diastolic, or a thrill may be felt. On *percussion* dulness is obtained if the aneurysm is sufficiently near the surface to influence the note. *Auscultation* gives little information of value.

Pressure Signs.—These are best classified by the structures affected.

Nervous System.—Paralysis of the recurrent laryngeal nerve is common, causing a husky voice and characteristic cough. In some cases pressure on the sympathetic causes inequality of the pupils (in some cases due to interference with the blood supply by one carotid) or flushing of one side of the face.

Respiratory System.—Tracheal tugging is fairly common. Pressure on the trachea may cause severe stridor and dyspnea. The signs due to pressure on the bronchi and lungs vary greatly, depending on the situation and size of the aneurysm. The striking point is the difference in the signs on the two sides of the thorax.

Circulatory System.—The heart may be displaced. There may be marked disturbance in the blood flow to certain arteries, giving a decreased or delayed pulse with lower blood-pressure, or in some cases stopping the pulse wave entirely. Obstruction to the venous return causes dilatation of the veins of the head and neck, arms or thorax. With this there may be clubbing of the fingers.

Esophagus.—It is not wise to prove the occurrence of obstruction.

Bones.—The ribs may be eroded with a resulting visible tumor. With erosion of the vertebræ there is pain of maximum severity.

Treatment.—Before deciding as to the treatment of a patient with aortic aneurysm, the problem of the patient and the exact character of the aneurysm should be studied thoroughly. Is the aneurysm of a form which can permit of complete healing by clot formation? Evidently this is possible only in comparatively small sacculated aneurysms and is out of the question in those cases which involve a considerable portion of the aortic wall. We can hope for cure in a very limited number; for the majority palliative treatment is the best possible. The patient should be studied so that danger of injuring him by too drastic treatment is avoided. It is not rare to see patients in whom much harm has been done by too vigorous treatment.

If the aneurysm is small, apparently sacculated and the patient's condition is such as to justify it, an attempt at complete obliteration of the sac is worth a trial, but this demands so much on the part of the patient that few have the resolution to continue with it. The strict measures suggested by Tufnell offer the best chance for obliteration of the sac. The few patients known to the writer in whom complete obliteration of the sac occurred (proved by autopsy after death from another disease) followed a rigid course. The principal points in this régime are:

Rest.—This has to be absolute, the patient not being allowed to do anything for himself. He has to lie absolutely quiet. So far as possible mental rest should be added to that of the body.

Diet.—The dietary advised by Tufnell contained 10 ounces of solid and 8 ounces of fluid. Difficulty comes particularly with the amount of fluid and it is rarely possible to keep to this small amount. But the closest possible approximation to it must be regarded as advisable. The amount should be reduced gradually until the minimum possible for the patient is found. As much variety as possible in the food given is advisable, and milk-toast, cereals, soft cooked vegetables and stewed fruits may be given.

Bowels.—These should be kept freely open so that straining is avoided, but with the restricted fluid intake this may be difficult. It is usually best to give differently acting laxatives. Mineral oil is useful in full doses with cascara or senna. The use of salines is advisable and in fact was an important part of the rigid Tufnell treatment as it drained away fluid. A full dose of Epsom salts every second day is advisable. It is hard on the patient perhaps but this is not an easy treatment and the patient must be prepared for it.

Medicine.—*Potassium* or *sodium iodid* should be given for a long period. The dose need not be large, beginning with 0.3 gm. (gr. v) three times a day and increasing gradually to double this amount. It is doubtful if anything is gained by larger doses. *Sedatives* may be required for the relief of discomfort or pain. For the former *sodium bromid* may be employed in the usual dosage, 1 gm. (gr. xv). For pain small doses of *codein,* such as 0.016 to 0.032 gm. (gr. ¼ to ½), may be given. Some patients do well on small doses of opium. For severe pain morphin, 0.016 to 0.032 gm. (gr. ¼ to ½) should be given hypodermically. The specific treatment should be given as described later.

The duration of this rigid treatment must depend on the results. A minimum of six weeks is desirable. If at the end of this time there are no signs of improvement it is not likely that a longer course will be useful but if there is decided decrease in expansion longer time should be given, say up to three months.

THORACIC ANEURYSM

Treatment.—For the vast majority of patients with thoracic aneurysm the best we can hope to accomplish is to prolong life and reduce suffering. In doing this there are certain measures which are of general application.

Rest.—This should be suited to the patient but usually a period of fairly rigid rest is advisable at first. This need not be absolute and the patient may be allowed up to void and defecate. He may be propped up in bed if this gives comfort and after a time allowed in a chair. After some weeks he may be permitted to increase his activities very gradually.

Diet.—In general this should be kept down to a reasonable minimum and in the majority of patients a loss in weight is an advantage. Some patients do well on milk diet which may be given for periods of a week at intervals. The amount of fluid should be restricted to about 1,000 c.c. per diem, and the solid food should be simple in character.

Bowels.—These should be kept freely open by the use of mineral oil, cascara, senna or phenolphthalein, with salines when required. Enemata are to be used if necessary.

Medicine.—The same treatment as regards iodid and sedatives should be carried out as already mentioned. The question of specific treatment always arises. In general it should be given, but always cautiously, remembering that the best results for each patient should guide us. It is well to begin with mercury by inunction, using 2 gm. (ʒ ss) of the ointment daily. If the general condition suffers or there is renal irritation this should be reduced or stopped. The writer's opinion is against the routine use of arsphenamin. If one of these preparations is to be given neo-arsphenamin is advisable, and the first dose should be 0.1 gm. If this is well tolerated the next dose may be 0.2 gm. but this should rarely be exceeded. Further dosage must be decided by the results. If no ill effects follow, regular alternating courses of mercury and neo-arsphenamin may be given.

Vasodilators.—In some cases these seem to be useful, sodium nitrite (0.03 to 0.06 gm., gr. ½ to 1) usually being the best. In some cases the more rapid effect of nitroglycerin may be useful for attacks of pain.[1] The value of vasodilators is sometimes seen in patients with low blood pressure. If the pressure is high the giving of the tincture of veratrum viride ♏ iv to viii (0.25 to 0.5 c.c.) is sometimes of value.

Sedatives.—These are usually required and while the main effect of iodid is relief of pain, other drugs may be required. Codein and morphin are the most useful drugs and should be given freely. . The dosage has to vary with the patient and in some patients with eroding aneurysms enormous amounts of morphin may be required.

Symptomatic.—The relief of pain has been mentioned. Patients sometimes come under observation with extreme distress and dyspnea. In these cases one remedy is of the greatest use—*venesection.* There should not be any hesitation in doing it, the quantity of blood withdrawn being decided by the results. Sometimes the withdrawal of a small amount gives immediate relief. It is usually well to give morphin hypodermically at the same time.

Surgical Measures.—These are used with the hope of promoting clotting in the sac. The most useful procedure is a combination of wiring and electrolysis. The method of wiring was introduced by Moore in 1864 and the addition of electrolysis was suggested by Corradi. This procedure has been very successful in the hands of my colleague, Hobart Hare, who has done the operation thirty-six times without any accident. He emphasizes the importance of using a fine gold platinum wire without too much spring (silver wire is not suitable), of using the positive pole in the aneurysm and of not giving too strong a current. This should be of 5 ma. at the beginning, gradually increased to 50, and then decreased to five, the current being passed for about fifty minutes. The wire is passed from a spool into the aneurysm through a hollow insulated needle, an average length of 30 to 40 feet being introduced. When the proper length has been introduced the current is applied, the external end of the wire being attached to the positive pole of the battery. A large clay electrode is attached to the negative pole and placed under the back. After about fifty minutes the wire is cut and the needle withdrawn. The most striking result is the relief of pain which may occur before the operation is finished. In some cases there is a marked decrease in the size of the aneurysm. Naturally the most suitable cases are those in which the aneurysm is sacculated, which can usually be determined by the X-rays. If the operation is not curative it nearly always causes marked improvement and may add greatly to the duration of life. One of Hare's patients lived for nine years after this treatment.

---

[1] Erythrol tetranitrate in doses of gr. ½ three times a day has a more lasting effect than the nitrites and agrees well with some patients.—Editor.

Of other measures, Macewen's operation of needling the sac has been useful, in some cases at least, but the risks are considerable. The method of occlusion by metal bands may prove useful in the future. In aneurysm of the abdominal aorta it may be possible to employ the brilliant method of Matas, so successful in aneurysm of smaller arteries, in an occasional suitable case. This consists essentially in controlling the circulation above the aneurysm, opening up the sac, cleaning out the clots, closing the opening into the artery, and plicating the outer coats over this.

Heart.—These patients often have myocardial disease which requires its proper treatment. There should not be any hesitation in giving digitalis, if its use is required, because the patient has an aneurysm.

Subsequent Management.—After any kind of treatment in which the condition is improved and the patient able to resume partial or fairly complete activity, he should be given very careful instruction as to his method of life. Overstrain and severe exertion should be avoided and if the patient has a laborious occupation he should change it for easier work. The hours spent in bed should be increased and a period of rest in the day is advisable. The diet should be kept down in amount and the bowels freely open. The giving of potassium iodid is advisable and the wisdom of the occasional administration of mercury should be decided by the results. Nothing should be done which lowers the general health. One patient under my observation came back every six months for venesection which he declared did him more good than any other therapeutic measure. It is probable that many patients with aortic aneurysm would be benefited by an occasional bleeding.

CHAPTER XIX

THROMBOSIS, EMBOLISM AND PHLEBITIS

Harlow Brooks

THROMBOSIS

Thrombosis, to a considerable degree, is a physiological process and plays an important rôle in the limitation of inflammatory processes, in the isolation of foreign bodies, in the checking of hemorrhage and in the spontaneous cure of embolism and phlebitis. It may also be one of the most serious pathological processes in any of the conditions mentioned, for it leads very frequently to embolism, to phlebitis, to pyemia or septicemia and it is one of the most difficult processes to control from either surgical or medical standpoint, in so far as treatment is concerned. It must be remembered that though all the chemical changes taking place in the blood in thrombosis are physiological and curative in intent, when pathological factors are superadded very grave lesions follow. It may be taken as an axiom that normal blood in a normal channel will not undergo thrombosis, but when the blood is altered in its characteristics, particularly when the clotting time is abnormally lessened, or when the walls of the vessel are diseased or traumatized, clotting will take place. When both these factors are active, thrombosis is the more certain to occur. Thrombosis plays a particularly active part in the various infectious diseases, either local or general. It is notably prevalent in typhoid fever and in pneumonia, and it may occur in any general infection. It is frequently associated with entirely local lesions, as in many surgical conditions, and it is particularly dominant in marantic states of all sorts and in many of the hemorrhagic diseases, in which possibly it may have a physiological intent.

Thrombosis extends by continuity or by embolism. Change in the intima of the blood-vessels is notably inductive of it and such general conditions as hemorrhage predispose to it. It may develop either as a cause or as a sequence of lymphangitis. In such general disease conditions as typhoid fever it appears to be invited, especially by stasis of the circulation, particularly in the capillary fields. This is undoubtedly a very prominent factor in the production of the thrombosis of the lung vessels in pneumonia and in mesenteric thrombosis. Torsion and indiscreet exposure and bruising of the tissue in operative procedures unquestionably predispose to the appearance of thrombosis, and surgeons are now giving more and more attention to these factors in all operations.

Treatment.—This must take into account all of the factors productive or provocative of the condition. Prophylactic measures have a real value beyond doubt. These consist chiefly in attempts to lower the coagulability of the blood. This may be accomplished by the administration of water in abundance, by the use of ammonia given in large doses and particularly by the giving of citric acid, either by stomach or by intravenous injection. Such measures are of particular value in cases of typhoid fever, pneumonia and the like in which a known tendency toward thrombosis exists, particularly when operative measures are contemplated. In cases of phlebitis, sinus thrombosis or embolism there is no doubt that the tendency toward thrombosis can be lessened by the use of such measures.

In all cases in which thrombosis is apprehended or feared, particularly if operative measures are likely to become imperative, especial care should be taken in the handling and transportation of the patient. This was particularly evident in the World War, where it was found that prolonged or delayed transportation favored thrombosis, especially in injuries in which extensive crushing of tissue had been inflicted. Great care must be exercised in such instances in the application of splints or bandages, so that too much constriction does not take place, especially in case of long evacuations. Wounded men should be given water very freely, especially after hemorrhage, all unnecessary cold and exposure must be avoided, and in the application of the tourniquet care should be taken to bruise the tissues as little as possible. In the removal of this instrument care must be exercised that clot is not swept off into the vessels, especially when infection is present. Food should be supplied whenever possible especially hot soups or broths, coffee or tea, whisky, brandy and wine, especially in shocked individuals who are particularly prone to thrombosis. In all operative procedures in these cases the surgeon must exercise as much care as possible to isolate contused and thrombosed tissues before excision is practiced. Where transfusions are necessary they should by choice be performed some time before the operation or immediately after, rather than immediately before, operation.

Some surgeons recommend that passive movement, massage and other activities be practiced in order to stimulate the circulation of the blood, but the majority of surgeons and physicians counsel, on the contrary, as near absolute rest as possible. There can be no doubt that thrombosis is invited when in the course of operation constricting apparatus, bandages and splints are applied as is the case in so many orthopedic, genitourinary and gynecologic operations. Prolonged fixation of an extremity in a cramped or unnatural position when the patient is on the operating table is to be avoided, nor does the modern surgeon advise, as in the past, the stripping of extremities and tight bandaging in order that the blood content of the trunk be raised, even when the extremity shortly is to be sacrificed. Transfusion is a much safer and more beneficial procedure.

After thrombosis has developed, if the location of the process permits, and if a collateral circulation has been satisfactorily established, ligature above and below the point of thrombosis is to be considered and removal of the area of diseased vessel is very frequently necessary, especially in sinus thrombosis. Great discretion must be exercised before taking this step and proof of a satisfactory subsidiary circulation established. This method of excision is particularly advocated when infection is known to be present. This measure is, however, often very debatable, especially in thrombosis of the mesenteric blood-vessels, and many surgeons feel that a better chance for recovery of function and saving of life is furnished by delay and by supplying facilities for adequate drainage. Chilling of the exposed tissue is especially to be avoided in all cases of thrombosis and a normal, or as nearly normal as possible, central circulatory activity is to be maintained, except in instances of cardiac thrombosis, such as appears particularly in endocarditis and in auricular fibrillation.

Pain in all circumstances must in so far as possible be controlled, both because one thereby promotes rest physically and because of the necessary general effect of rest in this condition. Sinclair recommends favorably electric ionization of potassium iodid with a constant current over the thrombotic areas. He believes this expedites a restoration of the disabled circulation. There is little doubt that the administration of chemicals designed to lessen clot formation is equally beneficial in a curative as in a prophylactic way. Jacobs reports a case of phlegmasia alba dolens in which the administration of ʒ I of citric acid dissolved in a glass of water, given three times daily, apparently brought about prompt and satisfactory results in limiting and dissolving the thrombus. Some clinicians recommended the use of the salts of citric acid by preference. More direct results are probably to be attained by the intravenous administration of the drug, but the clinician must also remember the long-established efficiency of water and of ammonia in accomplishing this result. It goes without saying, of course, that every effort, surgical and medical, must be exerted to support the general resistance of the patient, as by the use of iron, arsenic or of adequate food and drink, sun exposure and the like. In many cases the alkalis seem to promote resorption of the clot and the iodids, particularly in long standing cases, are of very certain benefit.

In apparently cured cases the greatest possible care must be instituted in a return to normal activities. The writer advises against massage and passive movements, rather depending on the caution of the patient who is usually solicitous to avoid a return of the process, and who is usually more acutely alive to any tendency toward overactivity than any person more detached can be. The writer's mistakes have chiefly consisted in a too rapid return to normal activities.

Cardiac Thrombosis.—Though considered under another heading, a few words as to the treatment of cardiac thrombosis are not out of place here. In most instances, the condition is chiefly caused by cardiac, notably auricular, dilatation with more or less cardiac stasis. The diagnosis is most difficult, especially when associated with a septicemia, and is pregnant with danger because of the likelihood of embolism at any time. It is possible that the measures so frequently mentioned, designed to lower the coagulation of the blood, may also be of value in this condition: however, this is extremely problematic and beyond doubt the most important therapeutic measure is to favor cardiac inactivity in so far as possible until the thrombus has either been resorbed or until it has become thoroughly organized. This may occasionally take place, as most pathological anatomists are aware, to a quite remarkable degree and the rapidity of organization may be surprisingly great. The chief source of danger is in the fresh layers of clot which are laid down on the old while the more parietal zones are undergoing organization. When the condition is suspected, and it should be in most cases of auricular dilatation, especially when associated with infection, the heart should not be stimulated beyond the point of necessity and cardiac rest rather than stimulation should be the goal. Recent experience seems to indicate that quinidin, through its effects on the auricle, is particularly likely to cause the dislodgment of thrombi which may then embolize perhaps in vital centers. Digitalis, in the opinion of the writer, if pushed to its full therapeutic or physiological effect, may produce the same change. Rest is the keynote of treatment.

Pulmonary Thrombosis.—Pulmonary thrombosis is an extremely frequent condition, far more so than is often realized by the clinician. It is a frequent accompaniment of pneumonia of all types, it is common in typhoid fever and in most marantic conditions and it develops as a natural sequence of not immediately fatal pulmonary embolism in practically all instances. Apparently citric acid is quite as useful in this condition as in any other form of thrombosis. Rest is of primal importance and time is the greatest curative agent of all. Oxygen inhalations are recommended by some, but the writer is not enthusiastic as to their beneficial results. The usual expectorants, especially the salts of ammonia and the iodids in the latter phases of the condition, are very useful. Artificial pneumothorax is contra-indicated in the experience of the writer, though favored by some. The favorable result implies in most cases organization of the infarcted lung tissue, but with frequent development of bronchiectasis and of lung abscess.

Mesenteric Thrombosis.—Surgeons are still greatly divided as to the correct method of treatment in this condition. It originates most frequently in peritoneal abscess and other intraperitoneal infections, occasionally from embolism. It is especially frequent in gangrenous appendicitis. There is no doubt that complete surgical removal of the thrombosed vessels with the dependent portion of the intestine would be the ideal treatment, but, in many if not most instances, this would be almost certainly fatal. In many instances also such complete removal is technically impossible or impractical, and the necessary technical traumatism inflicted in the operation would tend to disseminate rather than limit the process. Most surgeons of experience seem to prefer establishing drainage and waiting for nature to effect the cure in its own way.

Peripheral Thrombosis.—This condition is practically always a thrombophlebitis and for that reason the details of treatment are considered under phlebitis.

EMBOLISM

Embolism occurs from the presence in the blood-stream of bodies too large to pass through the capillaries. When these bodies meet a lumen not permeable to them they lodge and produce a stoppage of circulation at that point. Embolism is essentially a mechanical process, but it is succeeded, and in many instances preceded by thrombosis. The acute symptoms are produced directly by the mechanical effects of the embolism; subsequent signs and symptoms are due to and largely dependent on the nature of the embolus. For example, when it consists of infectious material pyemic symptoms may appear from the thrombosis which succeeds the embolism. The immediate signs and symptoms are caused by the embolus, the succeeding train of symptoms and signs are the direct sequence of thrombosis which is then indissolubly associated with the problem of embolism from whatever process it may have originated.

Embolism is brought about most frequently as a secondary process to thrombosis, arteritis or phlebitis. It occurs also with considerable frequency from the dislodgment of bits of material originating from an endocarditis or a cardiac thrombus. It may appear when, for any reason, air or gas enters the blood-vessels as in gas phlegmon, in caisson-disease, or in extensive traumatisms, especially in shell wounds or in other extensive lacerations of tissue. It is occasionally seen after surgical operations, particularly those involving the large venous trunks, and it infrequently develops in emphysema of either mechanical or infectious origin. Air embolism is less common in man than in many animals.

Fat embolism may occur when fat enters the circulatory channels. It appears in extensive injury of bone marrow, particularly in crushing trauma of bones when the veins are opened, and also when injury of fatty tissues takes place either in traumatism or in surgical procedures.

Resembling in some respects fat embolism is that which occasionally appears in fat necrosis, especially that form prevalent in pancreatic disease or injury. Another infrequent but pathologically very interesting form of embolism occurs from detached tissue when extensive crushing injury of soft viscera with laceration of veins appears, notably seen in traumatic injury of the brain or liver. Embolism also occurs with considerable frequency in various cachectic conditions as carcinosis, Addison's disease, pernicious anemia and the like. The probable mechanism in these conditions is a primary thrombotic process which may occur, as Conner has shown to be the case in typhoid fever notably, in fields of capillary thrombosis from which bits of detached thrombus are swept off either into the arterial or the venous circulation. It seems also highly probable that a similar mechanism is largely at fault in many postoperative embolic conditions, particularly in inflammatory conditions of the abdomen.

The symptoms and signs of acute embolism depend on the location of the embolism; on its extent, measured in the area of tissue normally supplied by the obstructed vessel; on the possibility of collateral circulation, on the secondary changes which follow the acute embolism and on the functional defects which appear either as a direct result of the lesion or from the secondary changes which follow it. An immediate result may be death if a vital center is involved. Secondary results are controlled mostly by the extent of the secondary thrombosis, by the degree of the collateral circulation, or by the subsequent absorption which takes place and such secondary lesions as may develop.

General Treatment.—After an embolism has taken place the treatment, save for certain immediate measures, resolves itself into the treatment of thrombosis and this has been considered under that heading. Obviously the treatment immediately demanded in acute embolism of any character depends chiefly on the location of the embolus and, of course, to a certain degree on the nature of the embolus. For this reason the above introduction has been given, for it is quite beyond possibility for the writer to consider therapeusis of any character unless based on a more or less definite picture of the mechanics or of the pathology concerned.

Preventive Treatment.—The preventive treatment of embolism is now entitled to a very definite place in our armamentarium. Acute embolism after surgical operations, especially in abdominal surgery, is so common that many surgeons are now attempting to take steps to mitigate its occurrence. Sinclair states that the danger of embolism from surgical procedure is greater than that which exists in phlebitis, even in phlegmasia alba dolens. Lockhart-Mummery makes the statement that the thrombosis which in so large a number of cases leads to embolism is favored by a slowed circulation. Some writers advise against the preliminary use of morphin in anesthesia because it is alleged to favor this. Several surgeons have recently advocated more postoperative activity than is customary, and they even urge the administration of such drugs as atropin or digitalis to accelerate circulation, and thus counteract a tendency toward embolism. These same writers in general advocate similar procedures in deciding as to postoperative physical activities. Thus James Gorton believes that he has limited the occurrence of embolism after abdominal operations by greatly decreasing the usual period of confinement to bed. He urges most of his patients undergoing abdominal sections, the general condition permitting, to leave bed and to even walk about within a few hours or days after operation. Early massage of the extremities, and active and passive movements with the same point of view are advised by some operators.

Equally experienced surgeons, however, advise absolutely opposite methods and urge prolonged rest in the belief that they thereby limit the occurrence of embolism by favoring the full organization of operative thrombi, thus lessening the liability to embolism occurring from them. There can be little doubt that in such extensive traumatism as occurs in war and industrial surgery, notably after high explosive contusions and wounds in which extensive blood-vessel injury has taken place, rest of the injured part, and rest and quiet of the individual definitely limits the liability toward embolism. It seems quite certain that unnecessary manipulation of the wound, long transportation and the like do increase a tendency toward embolism in such instances, and this is a factor well worthy of consideration in the great problem of the evacuation of the wounded. These same measures are of particular pertinence in the consideration of the prevention of fat embolism in conditions in which there has been wide traumatism and exposure of adipose or of bone tissue. There can be no doubt that the greatest gentleness and care in the manipulation of such patients will be rewarded by a lessened occurrence of embolism.

Lockhart-Mummery is of the opinion that the usual preliminary purgation, starvation and limitation of water in both preoperative and postoperative conditions, which has been and still is so very generally employed by most surgeons favors directly the production of embolism by increasing the concentration of the blood and decreasing the clotting time. He advises very strongly against these time-honored customs. He also urges the use of well-padded operating tables and the elimination of constrained and fixed postures both immediately after and during operations as of very definite utility in the prevention of postoperative embolism. The frequency of embolism after genito-urinary, gynecologic and gall-bladder operations is possibly attributable to the unnatural positions in which the patient is placed during these often prolonged procedures. He recommends that after operations the patient should be urged to move about and that the large muscle masses of the legs, thighs and buttocks be kneaded in order to promote venous circulation in the cava. Of course, these very procedures are considered to be of a particularly dangerous character for the same reason by many other operators, and the consensus of opinion is against it.

Practically all surgeons agree as to the need of gentleness of manipulation in all surgical procedures involving contused or highly vascularized tissues, and emphasize the advantage of the knife in preference to the cautery and mass removal in contradistinction to morselization. From this standpoint the control of hemorrhage by ligature rather than by compression is more desirable and is based on the same theory of embolus formation.

Surgeons are more and more coming to a serious consideration of the relation of operative technic to embolism. Of late in particular, thought has been directed to the frequency of pulmonary embolism as a result of crushing operations upon the tonsils. This does not take into account at all the advisability or inadvisability of general as compared to local anesthesia in tonsillectomy. The question at point here seems to be chiefly, if not exclusively, that of an aspiration bronchitis, abscess or bronchopneumonia with possible after effects.

Preparatory treatment against embolism may be practiced when the operation decided upon is one in which embolism is likely to occur. The coagulability of the blood may be lowered by the administration of citric acid, either by mouth or by intravenous injection. Water may be given liberally for the same purpose and excessive purgation should be avoided. Some surgeons give preparatory vaccinations with killed cultures of colon bacilli or with streptococci in the hope of thereby lessening the likelihood of infections with these organisms which are thought to induce after thrombosis embolism, especially in operations involving the intestinal tract. The writer has not seen evidence convincing enough to persuade him that there is actual merit in this procedure. In the operations which demand a previous blood transfusion it is apparently believed that less tendency toward embolism exists if the transfusion is done some time before the operation rather than immediately preceding or succeeding it.

In the course of necessary operations, especially where areas of thrombosis are known or surmised to be present, or where such conditions as an endocarditis are known to exist, as great care as possible should be exerted to obviate blood-vessel damage, as from tightly applied dressings, restraining apparatus and the like. In the course of the operation as little handling and damage as possible to the tissues in general, and especially to the highly vascularized ones should be made, and stripping of the extremities, as is sometimes practiced in amputations, should never be done. This last was found to be a particularly dangerous practice after the traumatisms and contusions met with in the recent War, especially in those cases in which the tourniquet had been applied. All inadvised injury to the blood-vessels should be avoided and the unnecessary application of ligatures must not be allowed, though at the same time cauterization or the checking of hemorrhage by the crushing of vessels is not favored. The general impression among surgeons also seems to be against the use of the styptics so long in vogue.

Whenever conditions favoring embolism are known to exist, such as a phlebitis, endocarditis, cardiac thrombosis and the like, as little stimulation or motion of the involved area as possible should be permitted. For example, in endocarditis, unless actual cardiac failure threatens, the use of such cardiac stimulants as caffein, digitalis and atropin are contraindicated. When dilatation of the auricles is known to be present, as in auricular fibrillation, the likelihood of auricular clot being great, such drugs as bring about a rapid auricular contracture with resulting dislodgment of clot are to be avoided in so far as possible. This has been shown to be particularly dangerous in the use of quinidin in auricular fibrillation but probably all physicians have had the same accident happen without any drug employment, or when other agents as digitalis or caffein have been used. Thrombosed extremities must be kept as quiet as possible and massage, kneading, etc., in old phlebitis are to be introduced most carefully, and only when it is believed that all thrombosed areas have been thoroughly organized.

It also goes without discussion that the whole body must be kept as quiet as possible whenever conditions known to favor embolism exist, and a release to normal activity must be accomplished cautiously. It is obvious that, in all operative procedures on the bone-marrow or on adipose tissue, careful ligation of efferent vessels should be performed before crushing of the fatty tissue is permissible. All surgeons realize the importance of avoiding air embolism in operating, especially upon or in the neighborhood of the larger veins of the neck and abdomen. It seems, however, that man is able to stand a pretty high degree of air or fat embolism, particularly as compared to certain of the usual experimental animals, notably the rabbit. Finally, every physician who attempts intravenous or even intramuscular medication should follow out implicitly the necessary rules for the prevention of embolism from foreign bodies of this nature. The writer has through carelessness caused embolism, fortunately never fatal or serious, from the injection intramuscularly of oily suspensions of mercury, and a good many instances of embolism from the use intravenously of arsphenamin and similar drugs are on record, some published and some not.

Treatment of Fat Embolism.—Buerger recommends that the immediate application of an Esmarch bandage will prevent further distribution of the fat already broken up by the blood-vessels and lymphatics. Rich particularly emphasizes the importance of care in the manipulation and transportation of all fracture cases. Wilms (quoted by Da Costa) treated one such case successfully by making a fistula in the thoracic duct. He provided seepage in incisions through fatty tissue by placing a provisional drain of rubber tissue in the angles of the wound, removing it after twenty-four hours. Patients with fat embolism should have absolute rest, particularly of the injured part. Free drainage of the part should be established, stimulants administered, and the use of external heat and the inhalation of oxygen urged.

Embolism of the Extremities.—The extremity involved must be placed in as easy a position as possible; one in which no muscular contraction or any unnecessary vessel pressure takes place. Pain is to be relieved just as promptly as possible by the use of morphin. Great care must be exercised that no unnecessary movement of the part is permitted and the patient's friends must be particularly warned against massage, kneading or any of the numerous manipulative types of treatment. Bandages and restraining apparatus must be applied loosely if at all, and when the extremity must be moved this must be done without the intervention of the patient's own muscle power. The extremity must be kept warm if the embolism is of a large trunk and if the peripheral circulation is nearly or completely obtunded. In this regard, however, great care must be exercised that no vesication of tissue takes place, and the writer has found for this purpose that the best results are accomplished by dry electric or natural sun heat. Hot-water bags are very dangerous, especially as more or less trophic and heat sense disturbances are almost certain to be present. The skin may be kept moist if necessary by non-irritating oil preparations.

There is now a distinct tendency among surgeons to advise embolectomy and favorable results are being reported with considerable frequency. P. Bull advises that, if the clinical symptoms and signs of insufficient collateral circulation do not subside after three or four hours, operation must be seriously considered. Where the collateral circulation has been insufficient for a period of twelve hours, no favorable results following embolectomy have as yet been reported. Bull further states that when doubt as to the necessity of the operation exists it should be done, for if it fails the ligature furnishes no worse condition than an amputation which is apparently inevitable if nothing else is done. When the operation is performed every possible technical precaution should be observed to avoid unnecessary tissue and especially vascular injury, and thus prevent the development of thrombosis or further embolism.

Amputation is, of course, the alternative in complete embolism of main arteries in the extremities and the choice of time for operation should be determined in large part by the extension of thrombosis and the character of the gangrene which may appear. Of course, the operator should not sacrifice tissue or function for the purpose of saving time, but there are many instances in which loss of time spells great danger to life. This is particularly true in cases of nephritis, in the diabetic and the senile, instances of which have occurred with particular frequency in the practice of the writer. In the main he has regretted delay rather than haste in operation in cases of this character though amputations have become necessary from time to time. It must be remembered that the danger of gangrene is much greater from embolism than from ligature at the same point. Either with or without surgical procedure, when any systemic disease factor is present as diabetes, syphilis or gout, treatment for the general condition must be instituted together with the mechanical procedures.

Aortic Embolism.—This rare condition is fatal in 92 per cent of cases according to E. Hesse. The writer has seen 2 cases which survived and several others found at the postmortem table, but undiagnosed clinically. Early death before diagnosis is possible, is the rule, but cases which survive a week or more may be readily diagnosed. Hesse urges early aortotomy and removal of the embolus by the transperitoneal route, assuming that the embolism is lodged in its usual point, at the bifurcation. Aside from this measure, which is hardly possible to accomplish unless the embolism is located at the bifurcation and which at the best is in itself accompanied by great danger, rest is the only other possibility. We hope for the establishment of a collateral circulation with a possible later canalization of the thrombus which has formed at the point of embolism. This was the course followed in both healed cases observed by me; in neither, however, was the primary occlusion of the vessel probably complete. This is doubtless the condition in many instances of aortic embolism and it is the subsequent thrombosis which usually plays the leading rôle. When the collateral circulation fails, amputation of one or of both thighs is indicated and to obtain a successful result it must be performed early.

Cardiac Embolism.—Cardiac embolism is of two chief varieties. In the one, actual plugging of a heart valve may occur from the detachment of a thrombus of the polypoid type which has formed on the surface of the valve or from the lodgment of a ball thrombus, either in a valve orifice or obstructing one of the great afferent or efferent vessels. For the most part these conditions occur only in premortem states, are of little clinical interest, and are found only as pathological curiosities. Plugging of the aorta and pulmonary arteries in premortal conditions is not so rare, particularly in myocardial disease or in marantic conditions in their terminal phases, but there is no treatment of any avail and it is a condition of interest almost exclusively to the pathological anatomist. The same applies to the interesting paradoxical emboli of Zahn.

Embolism of the Coronary Arteries.—This is a frequent condition and, in the experience of the writer, primarily more frequent than coronary thrombosis, though, of course, thrombosis occurs subsequent to the embolism in every instance not immediately fatal and is usually the more important process in so far as symptomatology and outcome is concerned. The treatment of this condition is considered under the heading of Cardiac Diseases, but the general outlines must be mentioned here. Rest is the all-important factor and is to be accomplished by placing the patient in that position most comfortable to him, usually in a half-reclining posture, supported on pillows or a bed-rest. Dyspnea is usually much relieved by this posture. The pain in the condition is often excruciating, and is rarely relieved by vasodilators, such as the nitrites, benzyl benzoate and the like. The early administration of morphin is necessary. Cardiac stimulation is contra-indicated unless the patient shows signs of dominant cardiac decompensation or of cardiac rupture, when, of course, all manner of emergency medication designed to stimulate and perpetuate cardiac contractures is justified. The subsequent management, extensively discussed under the general heading of Cardiac Disorders, is that of the secondary, but more important, coronary thrombosis.

Pulmonary Embolism.—Pulmonary embolism occurs commonly in cases of endocarditis, of auricular thrombosis or in premortal conditions in which cardiac thrombosis is present. It is frequent in phlebitis, after operations of all kinds and in marantic conditions and general disease. Many cases are immediately fatal, the patient apparently dying largely as a result of the shock consequent upon pulmonary collapse. If the patient, however, survives the first twenty-four hours, recovery becomes increasingly more probable as time elapses, but the aspects of the case and the treatment now take on the general character of that in pulmonary thrombosis, which is discussed more fully under Pulmonary Diseases and is also mentioned under Pulmonary Thrombosis. The writer believes that the use of ammonia in large and frequent doses, recommended by Richardson, is the most satisfactory emergency treatment of this acute condition. While it lessens the liability to thrombosis through its influence on the formation of blood-clot, it also acts as an excellent general stimulant and it possesses this notable advantage over citric acid which is also indicated in the condition. Artificial respiration either with the pulmotor or by the Sylvester method is indicated, together with the use of adrenalin, strychnin and, in instances of serious cardiac embarrassment, of strophanthin, and the intravenous administration of digitalis. Mental and physical rest are of course necessary in these cases.

Cerebral Embolism.—Rest again furnishes the greatest essential in this condition, and by the administration of citric acid we are probably able also to limit the subsequent thrombus formation. Stimulation is usually unnecessary except where the embolus has lodged in some such vital center as the respiratory, when the appropriate symptomatic treatment must be observed. The subsequent treatment, after prolonged quiet, both physical and emotional, is best accomplished with the iodids and that of the consequent paralysis, if present, by massage, passive and active movements, electricity and the like. The writer has been strongly impressed with the prophylactic value of quiet and rest in all those conditions in which cerebral embolism is apprehended and in his opinion the vasodilators and morphin or other sedatives are of real value in the prevention of this condition.

Splenic and Renal Embolism.—These are extremely frequent and are very often clinically unrecognized because of the few symptoms which appear, aside from those of the immediate acute onset. There is no immediate treatment demanded except rest and the relief of pain by the prompt administration of morphin. In the case of renal embolism the subsequent treatment is that of a crippled kidney associated with physical rest and attention to such complicating conditions as may follow.

Embolism of the Mesenteric Vessels.—This condition is found much more frequently by the pathologist than it is diagnosed by the physician or surgeon. It develops usually in inflammatory processes of the abdomen, particularly in abdominal or pelvic abscesses, gangrenous appendicitis and the like. The pathological anatomist finds postmortem instances in which healing has apparently taken place followed by quite extensive thrombosis, without producing any very striking symptoms except occasionally those of intestinal obstruction. It occurs also with considerable frequency in or after operative procedures on the intestine and if the embolus be sterile it may undergo spontaneous cure. If, however, associated with infection, the condition is very likely to excite a spreading mesenteric thrombosis of very serious character. So excellent a surgeon as Albert Berg recommends that in a large percentage of these cases operative procedures be not undertaken. At best the mortality rate is very high. The general treatment for thrombosis may be instituted, and in suitable cases operation with careful resection of the involved tissue be recommended, but there are no very definite indications for treatment other than expectant and symptomatic measures.

PHLEBITIS

In point of frequency phlebitis is probably the most common of the three conditions under consideration. As in embolism the process is quite inseparably associated with that of thrombosis; indeed thrombosis plays a major rôle in all cases of phlebitis, so that complete separation is neither possible nor desirable, no matter from what angle one wishes to consider the subject.

Curiously, phlebitis is mostly a disease of the peripheral veins. In point of occurrence it is infrequently found in the veins of the viscera as compared to those of the extremities and in most instances when it occurs in the viscera or in portions of the trunk it is a secondary process, or an extension from phlebitic foci originating in the extremities. Again, in the extremities it is far more common in the superficial than in the deeper tissue layers. It is notably frequent in all its types in the veins of the legs and thighs, especially in the internal saphenous and its tributaries. It also appears very frequently in the veins of the upper extremities. From these trunks it extends to the vessels of the pectoral regions, to the veins of the mammæ, or, in case of the lower extremities, to the veins of the abdomen.

Although this definite selective character, that is, to involve chiefly certain trunks is very manifest, none the less in terminal and hemic states, and in others in which a tendency toward thrombosis exists, it has a far more determining influence in the disease than any conditions which primarily exist in the walls of the veins themselves. It is comparatively infrequent even in extensive crushing injuries that phlebitis of the veins of the extremities develops when infection does not exist, or when unaccompanied by hemic conditions which in themselves tend to induce thrombosis. The extensive experiments of Vaquez especially demonstrated this fact, which is further borne out by general clinical experience. When, however, a tendency to thrombosis already exists, traumatic sterile injury of the veins tends very definitely to invite and determine the location of extensions. This is a clinical fact which must never be lost sight of in the management of the disease. The same general principles pertain in phlebitis as exist in thrombosis and embolism, but they need not be considered in a text dealing primarily with problems of treatment.

Thrombosis plays the dominant part in every instance of phlebitis. Usually, however, a phlebitis does not arise in a thrombosed vein except as a reconstructive process, unless the added factor of infection or of some other irritant is present. When infection is introduced from without, as in a periphlebitis, and extension through the walls takes place, thrombosis then develops. Such instances occur when the vein courses through inflammatory tissues, as in infected wounds, abscess cavities and the like. To some extent the same end result follows when the infectious elements are absent or of a lesser degree, when the walls of the vein are roughened, as in phlebosclerosis. This is entirely comparable to an endarteritis, though much less frequent. When, due to either general conditions or to local disease such as typhoid fever or syphilis, the vitality or normality of the vein walls is reduced, phlebosclerosis is far more likely to follow.

The histological changes which take place in the walls of the veins in phlebitis are precisely similar to those which occur in inflammatory conditions elsewhere. These are an infiltration of small round cells, a proliferation of the connective tissue cells and the formation of new capillaries which grow into the thrombus. This then may become organized and finally transformed into a rod of scar tissue into which a deposit of lime salts may take place thus forming a phlebolith. When, however, infection is present, necrosis and liquefaction may take place forming virtually an endovenous abscess. From this focus, infected clot or pus may be swept off into the general venous circulation, perhaps to be carried upward into the large trunks, through the right chambers of the heart and thence may perhaps lodge in the radicles of the pulmonary vessels, thus causing pulmonary embolism and infarction. Connor has called particular attention to the frequency of this process in the phlebitis of typhoid fever, and he has apparently demonstrated that minute and clinically unrecognized phlebitis in minute venules may thus cause pulmonary embolism of very important grade.

There can be no question whatever that mechanical activity of any thrombosed part, as in a phlegmasia alba dolens of whatever origin, tends to favor the dislodgment of these minute thrombi. The natural deduction is, therefore, to emphasize the importance of absolute rest in any case of phlebitis, and of local rest of the part in which the phlebitis is present. The great danger of unsupervised massage or other mechanical measures, even in apparently healed cases of phlebitis, is brought out.

General septicemia, multiple embolism and thrombosis and, depending on the character of the infecting agent, pyemia may thus follow a phlebitis.

Phlebitis is much more likely to involve those veins furthest from the heart, probably because of reduced rapidity of the blood-stream there. Partly also as a result of gravity itself, thrombi causing phlebitis are most likely to occur in the extremities, notably in the lower ones. The process is probably also more frequent in these areas because of the greater frequency of mechanical injury such as may result in the handling of patients suffering from debilitating conditions as carcinosis and tuberculosis. Another pertinent reason for the very frequent occurrence of phlebitis in the veins of the lower extremities is the common occurrence in these parts of varicose veins, the walls of which eventually develop more or less phlebosclerosis and the varicose character of which induces a sluggish blood flow. This recognized tendency is still another argument for great care in the management of cases of varicose veins, particularly those of the pampiniform plexus and of the lower extremities.

Varicose ulcers of the legs notably and superficial varicose ulcerations anywhere are very likely to give origin to diffuse dissemination of septic thrombi, and to spread phlebitis. This fact must be fully considered by the surgeon in his operative management of varicose ulcers. Phlebitis also develops in the course of lymphangitis of any part, probably from a direct extension of the inflammatory process through the walls of the vein, particularly if associated with an infection.

For clinical purposes it is convenient to classify a phlebitis from its probable etiology or from its symptomatology, because the physician is thus directed more definitely toward a special management of the case designed to attack specifically the underlying basic pathogenesis. Sansom has presented a classification which seems particularly practical. To a very considerable degree I have followed his classification.

Non-obliterative Phlebitis.—This may be classed as a form in which complete obliteration of the involved vein does not take place or, if it occurs, it does not persist. Such cases are marked by the appearance of widely separated foci of phlebitis, in which the thrombosis is rarely complete, and in which venous stasis may not therefore become marked, but from which multiple foci appear often long after the primary process or infection has subsided. Such cases are sometimes classed as vagrant phlebitis. They develop in considerable frequency from varicose ulcers, usually of small extent, from postpartum infections, especially in their early stages, and they may also appear in syphilis, usually in the late stages. The symptoms in these cases are very often entirely out of proportion to the apparent degree of the lesions which are not as a rule either large or apparently serious. Fever may be high and is usually accompanied by a rapid pulse, high leukocytosis, marked prostration and often by chills and sweat. Pain in the involved area is usually sudden in onset and may be very severe. The lesion is not commonly that of a cordlike induration marking the line of the involved vein, but a spherical or ovoid area of inflammation, sometimes closely resembling the lesion of an erythema nodosum. These lesions may quickly and completely be resorbed and they often leave almost no detectable trace in the way of an indurated or sclerosed vein, but relief of one is usually quickly followed by the appearance of another. Sometimes they appear in crops and there seems to be a tendency in some instances to the symmetrical distribution of the lesions. In my experience staphylococci and the Streptococcus viridans are very frequently found in this type of phlebitis and endocarditis is a very frequent complication. These cases are, even in favorable instances, long drawn out and a fatal termination from endocarditis, septicemia, or from embolism is very frequent. Two cases of this type have, in my personal experience, recovered without any permanent disability.

Obliterative Phlebitis.—These cases are also usually embolic in origin and sudden in onset. They appear commonly with the symptoms of a sudden sharp pain in the involved area, quickly followed by the appearance of a cordlike thickening about the involved vein, local tenderness, pain and swelling. The area of induration persists, for a short time it extends, but always maintains its oblong or cordlike shape, except as it may invade adjacent anastomosing trunks. The symptoms and signs are usually definitely local, there is little fever, pulse acceleration, or other evidence of general disease. As the process recedes the induration becomes more definitely focalized in and about the involved vein which in healed cases usually persists as a definite cordlike structure. Dissemination, as in the form just described, does not take place as a rule, though of course similar foci may appear elsewhere, probably derived from the same original focus.

Sometimes the area may break down into an indolent ulcerative process which may strongly suggest a trophic ulcer. Occasionally in unfavorable cases quite wide extension to adjacent venous trunks takes place and sometimes one eventually sees quite extensive loss of tissue. The cases rarely, however, take on the picture of a general infection and, as a rule, the blood culture will be found negative. General symptoms and signs are absent, there is no leukocytosis, and though healing is very often slow and occasionally quite extensive disability may follow, the outlook on the whole is far more favorable than in the type just described. This form appears also with great frequency in postpartum cases and apparently it is frequently due to a sterile thrombus dislodged from the uterine sinuses. When the number of involved trunks becomes large and when the process is complicated by a secondary lymphangitis, the familiar picture of phlegmasia alba dolens is presented. This is also frequently the type presented in typhoid phlebitis. Its most frequent serious complication is a pulmonary or coronary embolism.

Recurrent Phlebitis.—This is a type of chronic phlebitis which develops most frequently in old cases of obliterative phlebitis. The lesions do not entirely clear up, but remain more or less tender. There is a tendency on the part of the superjacent skin to break down and ulcerate and there is a very definite tendency for adjacent veins to become involved. Perhaps due to obstructed return flow in the original area, other veins become varicose and slight bruises or injuries are very likely to excite a return of the extending thrombosis. Infection of the devitalized area from an eczema or slight skin abrasion is likely to set up a cellulitis or perhaps an erysipelatous inflammation of the area. This type of phlebitis is particularly likely to appear and to persist for an interminable period in the aged, tuberculous, gouty or syphilitic patient, and may become eventually a surgical condition in so far as its management is concerned. Amputations, notably in diabetics, arteriosclerotics, are often demanded, especially if outside infections be introduced. This form is very frequent in hospitals for chronic diseases and in homes for the aged. Early surgical care will prevent a great many of these cases from becoming serious problems. One cannot help feeling that in the majority of instances they result from an outside infection superimposed on some constitutional defect.

Chronic Phlebitis.—This is, as Sansom states, a "persistent painful or tender induration of the vein walls, resulting in atheroma." As a matter of fact it appears to be elementally a phlebosclerosis in most instances, and it is seen in the aged and infirm who have varicose veins, notably in patients who have suffered for a long time from gout, syphilis, nephritis, diabetes and long-standing tuberculosis. It is usually associated with more or less general arteriosclerosis, and is probably an analogous process affecting chiefly the veins. Thrombosis is likely to occur at any time in these cases, extensive disability of the member involved takes place and trophic change due to involvement of the nerve trunks is generally more or less present. Occasionally these symptoms and signs dominate the picture to such an extent that the case may be very readily taken for one of primary spinal or peripheral nerve disease.

So very familiar is the picture of phlegmasia alba dolens that it seems unwise to attempt description of the condition here, or to discuss its well-understood pathogenesis, pathological anatomy, signs and symptoms in a text designed particularly for the discussion of treatment.

Its occurrence is not limited to postpartum cases, but it appears also in typhoid, in pneumonia, and especially in influenza. It is of quite as much interest to the internist as to the obstetrician or general practitioner. Briefly it may be summarized as a phlebitis, usually of the obliterative type, associated with a lymphangitis of greater or lesser extent. Obstetricians see it mostly involving the lower extremities, and in typhoid fever the same is generally true, but in the other infections, notably pneumonia and influenza, the upper extremities are almost as frequently the primary site of the process. One fact must always be borne in mind by the practitioner, namely, that this type of phlebitis commonly develops rather slowly and that the patient may complain of pain long before signs appear. The pain is usually of a radiating character, and is usually ascribed to some group of muscles as the calf or thigh group. There may or may not be fever. If present it is not as a rule very high, nor of sudden onset. The pain may persist for a considerable time before other signs indicative of the disease are apparent. Frequently practitioners feel that the pain may be a neuritis, a myalgia or even an hysterical manifestation up to the time when edema, localized tenderness and induration, often deeply placed along a vein trunk, appear usually with more or less coldness and cyanosis of the peripheral portion of the involved extremity. The very great importance of remembering this insidious onset lies in the absolute necessity for early diagnosis and prompt application of treatment in this condition if satisfactory results are to be expected.

General Treatment.—Even before diagnosis has been verified and often before it is more than mere supposition, the suspicious extremity must be immobilized. The position to be selected for immobilization is determined by finding that pose in which the greatest relief from pain is given to the patient. He must be put to bed in every instance and he must from the first be cautioned against sudden and unnecessary movements and absolutely advised against massage, kneading, vibration and other mechanical measures. I have seen such measures, in several instances, especially in the early stages, directly dislodge a thrombus and produce fatal pulmonary embolism. If absolute quiet cannot be secured otherwise, the prompt use of morphin is certainly indicated.

After quiet in bed and a comfortable position for the involved extremity have been secured, care should be taken that no skin infections take place.

Most scrupulous supervision is often necessary in this respect, especially when the involved veins are deeply situated and of large caliber. Moist applications should never be permitted at this period of the process nor until some real idea has been obtained as to the degree of tissue devitalization which is likely to occur. As a rule, the extremity is most comfortably supported by a canvas sling covered by dry cotton. The surface of the skin must be gently wiped free from sweat and powdered with talc or zinc stearate. It must be carefully inspected daily so that no small abrasion or pustule may escape early detection and prompt treatment.

Heat properly applied usually gives considerable relief, but it must not be prescribed in a haphazard manner. A temperature above 110 degrees must be used only very carefully. The careless use of the hot-water bag or electric pad may do a great deal of harm, particularly in those frequent cases where wide involvement is present. Heat is best applied by an electric bulb introduced in a space produced by a tunnel or wire cage in such a way that the light cannot come into direct contact with the extremity. The limb should be wrapped in a layer of cotton or surrounded by a smooth, soft layer of blanket or gauze. Sinclair mentions favorably the use of electric ionization.

If the pain is very severe and is not controlled by the heat, I advise the use of aspirin, of luminal and, if not contra-indicated by a cardiac condition, of phenacetin or some of the other non-habit-forming analgesics. Codein or morphin must, however, be used where the pain is very severe, or when such soporifics as chloral, adalin, paraldehyd, or the bromids fail to make it possible for the patient to rest and sleep in relative comfort. Where the use of opium is imperative, I have found it advisable to vary the form as frequently as possible, thus tending to lessen the liability to habit formation. One can give it by suppository, by mouth or hypodermically, varying the form of the preparation so that the patient may be kept unaware of the drug used. Relief of pain and quiet are often imperative in these cases at almost any cost.

The patient must be put upon a light, easily digested but sufficient diet, the character of which will depend very largely on the character of the primary disease in which the phlebitis has developed as a complication. The general body resistance must not be allowed to fall or extension is favored. The bowels must be kept well opened, saline laxatives being usually the most satisfactory. Occasional colonic lavage is very beneficial. The possibility of increasing a tendency to thrombosis as a result of purgation must, however, be considered. The renal activity must be kept in mind and observed by frequent urinalysis and the character of the diet determined somewhat thereby. I see no reason for limiting the intake of either salt or water unless general edema or some other direct evidence of renal or circulatory inadequacy appears. In gouty or nephritic cases the diet must be modified accordingly. The blood value must not be allowed to deteriorate. Frequent determinations of the hemoglobin should be made and falls corrected by the administration of iron or, in suitable instances, arsenic. The physician must never neglect the general condition in his intensive treatment of the case, and frequent physical examinations should be the rule. The symptomatic management of the case is oftentimes of far greater importance in obtaining a satisfactory ultimate result than the specific care of the phlebitis alone, and the physician must never lose sight of an underlying general disease and such factors as may tend to eliminate further tendencies toward the development and spread of the phlebitis.

There can be no doubt that the use of certain measures does tend to lessen the liability of extension of the thrombosis. First among these, because simplest and most natural, is the administration of sufficient water provided other conditions do not contra-indicate this. There can be little doubt but that the use of citric acid intravenously limits the tendency to thrombosis. Jacobs reports very definite benefit in phlegmasia alba dolens by the use of sodium citrate given by mouth. He advocates giving 1 dr. dissolved in a glass of water three times daily. By this means he was able greatly to prolong the clotting time of the blood and the result in the case reported, so far as dissolution of the thrombus was concerned, seems to have been very definite.

Small doses of potassium iodid introduced intravenously have been favorably mentioned by several observers. They are apparently of greatest benefit in the latter stages of the disease, although they are supposed also to prolong the clotting time of the blood. Sterile ampules of the drug may now be obtained from various firms and thus far I have seen no unfavorable results from its use. In so far as mere clinical observation can go in a very limited number of cases, this method seems to be of real value, but the same result can apparently be reached by giving the iodid in the usual way, a method which I, in common with most practitioners, have long employed. I have felt that the iodids were particularly useful in cases of chronic phlebitis, or in those instances of thrombosis in which venous stasis was present primarily.

Sinclair has recently called attention again to the theoretical possibility of hastening clot consolidation and resorption by the giving of calcium salts. I do not advise this procedure, although experimental investigations conducted some years since by Crowell and myself have shown in rabbits how very difficult it is materially to alter the clotting time of the blood by the administration of lactate of lime. A much more definite tendency toward thrombosis is developed by reducing the body fluids by catharsis and withdrawal of fluid. This tendency must be given full consideration, especially when operative procedures are under consideration.

After the primary stages of the disease have passed and the degree of tissue injury or loss can be estimated, it is safe to introduce, except in diabetics, nephritics and advanced arteriosclerotics, warm to hot fomentations, oil dressings and such local applications as saturated solutions of magnesium or aluminum sulphate. These tend to reduce the edematous exudate and favor resorption. P. Bull advises the use of compresses saturated with 2 per cent sodium nitrite solution and suggests that in surgical procedures the surgeons' hands and instruments be rinsed with this solution in an attempt to minimize secondary thrombosis. I am much opposed to the use of such preparations where abrasions of the skin are present, for infection or moist gangrene may supervene. They no doubt hasten resorption in a good many instances and they often relieve pain. After all acute symptoms have subsided, gentle bandaging may be introduced, using for this purpose preferably the French crêpe bandages or those made of similar elastic but non-rubber-containing material. Very gentle and carefully supervised passive movements of increasing intensity may eventually be introduced, so that the muscle atrophy does not proceed too far, but it must be a long time before the patient is allowed out of bed or to use the involved extremity at will. Fortunately, the patient's caution in respect to movement and the resulting pain is sufficient to enforce the desired care, but a return to normal movement must be most cautiously approached. For a long time thereafter gentle bandaging of the part is advised while the patient must remain under medical observation usually for a much longer period than he believes necessary.

Quite frequently after phlegmasia alba dolens and also after other clinical forms of phlebitis, such serious nerve defects persist that some consideration of their management is desirable. In my experience this has occurred most frequently in patients with chronic phlebosclerosis, diabetes, nephritis, arteriosclerosis, gout, or syphilis. This result may follow in many instances from actual neuritis consequent upon direct invasion of the nerve trunks, or perhaps from the effect on the peripheral fibers of the toxic materials which bathe them. Most commonly of all in my experience it results from the encroachment of the fibrous tissue which follows the resorption of the inflammatory exudate. Doubtless also there are serious nutritional defects which take place in the nerve fibers. Great disturbance of sensation and of vasomotor control are often seen, and serious, even permanent, trophic changes may occur. The extent to which these lesions may mend themselves when sufficient time and persistent care is given them is often quite surprising, but medical and even surgical assistance is usually necessary. In several instances deformities have followed which have demanded tenotomies and very careful reconstructive surgery. This must, of course, be most advisedly considered before operation because of the likelihood of reëxciting either another phlebitis or an infection in the largely devitalized tissue. Carefully observed massage with stretching operations may be cautiously employed, but usually only after the patient has been well tested out on massage and active and passive movements. The iodids are of special use in cases of this kind. The use of diathermia or static and faradic electricity is highly recommended by some. Treatment with sunlight has also been employed with very definite benefit in cases under my observation. This usually possesses the great additional benefit of improving the general status of the patient, a thing which is always of primal importance, particularly in slowly convalescing cases.

C. de la Carrière speaks very enthusiastically of the treatment of all forms of phlebitis, except those due to cachectic disease, by hydromineral treatment, such as is employed at Bagnoles-de-l'Orne. He states that the hot mineral water cure is most efficacious when applied before the inflammatory exudates have time to organize. The treatment at Bagnoles-de-l'Orne consists of baths of which the temperature, always lower than body temperature, varies from 33° C. to 36° C. and of which the duration reaches from 50 to 60 minutes, these maximum periods being reached finally after primary short exposures. The results are said to be excellent.

Surgical Treatment.—Surgery has a distinct field in phlebitis as it has in thrombosis and in embolism. It is in some instances a safer and more beneficial procedure than medical treatment in so far as cure and removal of the condition is concerned. Cases occur, as in embolism, in which amputation of an involved extremity may be not only advisable but even imperative in order that the process may be limited, and that it may not extend to other territory. This is usually seen when thrombosis of an infectious character is present and also where embolism is either present or seriously apprehended.

Removal of a diseased vein may be demanded in some instances where pain is a dominant symptom; resection of the vein may give complete and final relief. As a rule, resection is not to be practiced as a means for checking a rapidly spreading thrombosis unless a collateral circulation has been pretty well established. The question of the surgical treatment of varicose ulcers comes into play in deciding these problems. In any instance, the surgeon must exercise great judgment and very considerable technical skill in deciding on the time for and the extent and character of the operation. He must constantly bear in mind the dangers of a spread which may follow extension of infection, thrombosis or disturbance of more or less already established circulatory paths. The technic employed must be thoroughly studied so that no undue traumatism is inflicted and at the same time great care must be exercised in the excision of all devitalized tissue. The poor nutrition of the tissues must be kept well in mind throughout. Extraordinary care to prevent septic processes must be exercised in all cases and mechanical traumatisms, such as may be inflicted with impunity in ordinary surgical procedures, must be minimized in the light of the known tendency to thrombosis, embolism and devitalization of tissue.

All that has been said as to the surgical treatment of embolism and thrombosis applies with equal force to surgical treatment in phlebitis, either of the acute or chronic forms. Sinclair makes the statement that thrombosis and embolism are much more likely to follow in postoperative phlebitis than in phlegmasia alba dolens, but does not advance any real explanatory data.

REFERENCES

Buerger. Quoted by Rich. Nebraska State Med. Journ., vii, 14, 1922.
Bull. Norsk. Mag. f. Laeger., lxxxiii, 137, 1922.
Carrière, de la. Presse méd., xxvii, 511, 1919.
Hesse. Arch. f. klin. Chir., cxv, 812, 1921.
Jacobs. Cleveland Med. Journ., ix, 441, 1910.
Lockhart-Mummery. Proc. Roy. Soc. Med., Surg. Sect., 15, 23, March, 1922.
Sansom. Twentieth Century Practice of Medicine, iv.
Sinclair. Brit. Med. Journ., i, 375, 1922.
Steel. Journ. Am. Med. Ass., lxxvi, 429, 1921.
Vaquez. Phlebite des Membres, Clin. méd. de la Charité, Paris, 1894.

CHAPTER XX

THROMBO-ANGIITIS OBLITERANS

S. J. Goldberg

Synonyms.—Presenile spontaneous gangrene, juvenile gangrene, endarteritis-obliterans, intermittent claudication.

Definition.—Thrombo-angiitis obliterans (named by Leo Buerger) is an organic, inflammatory, thrombotic disease of the blood vessels (arteries and veins), chiefly seen in the extremities but sometimes extending to the internal parts, in which there is occlusion of the lumen of sections of the vessel. This gives rise to impairment of function of the parts supplied by these vessels, or to complete incapacity of the extremity involved, loss of vitality, and finally gangrene.

Etiology.—No previous diseases seem to have any bearing as a direct causative factor in the production of this condition with the possible exception of syphilis. No true etiological factor that would fit in even with a small portion of cases has ever been presented. William Lusk of New York reported prompt and permanent improvement in several cases under intensive arsphenamine treatment. All his cases, however, showed a positive Wassermann test before treatment. Previous habits of smoking, mode and place of living, alcoholic habits, or the possession of an unstable and hypersensitive nervous system seem to have a direct bearing on the histories of the cases that I have seen.

Erb in his first description of this condition advanced the opinion that smoking was the most prominent etiological factor, and Willy Meyer several years ago discussed at great length the various toxins found in cigarette tobacco or generated by burning tobacco in any form. He even calculated the number of cubic feet of tobacco smoke laden with toxins a smoker of twenty cigarettes a day would inhale and concluded by putting the entire blame on tobacco, contending that the beginning of marked symptoms was coincident with the point of saturation of the body with these tobacco toxins. It is true that a large proportion of the patients suffering with this condition are inveterate cigarette smokers, and it is plausible that it might be a predisposing factor, but it will not explain the great majority of cases. I have had 6 cases out of a series of 30 whose tobacco habit was decidedly negligible.

In most of the cases living in Russia or Poland until adult life, one gets the familiar alcoholic history, always several small drinks of whisky each day because of the cold winters, but rarely intoxication. The history of the diet of these patients is characteristic, since most of them are Jewish, one notes that their diets are conspicuous for their excess of carbohydrates and fats. This fact suggests itself as being of extreme importance as an etiological factor producing this symptom-complex, because of the frequency with which high blood-sugar percentages (hyperglycemia) have been found by Bernhard and others.

Most of the patients belong to the poor or middle class, who for many generations, both in the land of their nativity and in this country, have undergone many hardships, physical and mental. Their housing has necessarily been very congested and often far from sanitary and, everything considered, not conducive to producing perfect health. It is well known that many of these people make up a conspicuous portion of that class known as psychoneurotics or neuropaths, an end product of many years lived day by day in constant fear of death, persecution or exile. But it is difficult to estimate how strong a factor this is in causing the disease.

Another element which is often put forth as an etiological or predisposing factor is the history of years of exposure of the extremities to severely cold weather, such as the winters of Russia, where many of these people come from. Such exposure to cold is common history in these patients, but other people in those countries suffer the same exposure and are not afflicted with this condition with any such marked frequency. It is also uncommon to find this condition in other races having occupations necessitating severe and continuous exposures, such as the lumbermen in the far north, longshoremen, truckmen, etc. It must be conceded that the true etiology has not been found although the elements mentioned might readily be predisposing factors.

History and Geographical Distribution.—For many years this disease belonged to the poorly defined group of symptom-complexes known as the vasomotor trophoneuroses. This group was made to include acrocyanosis, erythromelia, erythromelalgia and Raynaud's disease, because the clinical signs and symptoms in some stages are very much alike. But the extensive work of Leo Buerger and others, about 1908, showed conclusively that this disease was an actual thrombosis of the vessels, both arteries and veins, causing variable degrees of permanent occlusion. While Raynaud's disease and the others may for a time present the same symptoms and signs and may end in gangrene, the latter is caused entirely by a vasomotor neurosis causing variable degrees of spasm of the arteries and giving rise to intermittent and variable degrees of arterial occlusion.

The condition we are considering was described by several German clinicians as "Spontan-Gangrän" or spontanous presenile gangrene, as early as 1875, and a little later as "obliterating endarteritis." As early as 1879, von Winiwarter described the condition as an obliterating endarteritis caused by a proliferation of the fibrous and cellular elements of the intima, which process continued until there was a full occlusion of the vessel.

During the last ten years many cases answering the same description have been reported in Russia, Japan, China, and Persia. In this country it is quite commonly found in the large centers of population along the seaboard, particularly those harboring large numbers of Russians, Slavs and Poles, or their next generation. More than 600 cases are recorded in New York City alone. A fairly large number of cases have also been described in Wisconsin and Minnesota among the Slavonic settlers and the first generation of their descendants.

Incidence (Age, Sex and Race).—Thrombo-angiitis obliterans is a disease of the young and mid-adult age. In my series of 30 cases the youngest was twenty years of age and the oldest sixty years.

The enumeration was as follows:

Between 20 and 30 years	6 cases
Between 30 and 40 years	17 cases
Between 40 and 50 years	5 cases
Between 50 and 60 years	2 cases

The onset in many cases is so insidious and the course so slow, characterized by a tendency to periods of improvement, then relapses, that it is very likely that if a diagnosis could have been made in the initial stage of the disease we would find their ages really ranging some few years younger than stated. Willy Meyer is of the opinion that the disease begins shortly after puberty in many cases and gives rise to the characteristic symptoms sufficiently to be recognized only in early middle life.

All of my cases were in the male sex, except 1. The sex incidence given by those who have seen or collected many cases is about 1 female to 75 males. The most plausible reason for the rarity in the female sex is because of the repeated periodic congestion of the lower part of the body and lower extremities (as in menstruation and pregnancy), acting as a prohibitory measure.

By far the largest number of cases occur in the Hebrew race. The ratio would be even greater than that of male to female. The large majority of these cases are immigrants from Russia, Russian Poland or German Poland. If it is found that the patient is American born, it usually follows that his parents were immigrants. Naturally there are exceptions to this rule. About 75 per cent of cases were of the Jewish race. I have never seen 2 cases in one family.

Pathology.—According to Buerger, presenile spontaneous gangrene is not a protein manifestation of an arteriosclerotic process, but a distinct pathological and clinical entity characterized by an ascending thrombotic occlusion of the arteries alone, or of the arteries and veins. It begins usually in the blood-vessels of the legs below the bifurcation of the popliteal artery, and then spreads upwards to the larger trunks, or it may appear independently in other vessels. This occlusive thrombosis is followed by organization and canalization of the clot, and with the disappearance of the inflammatory process finally comes a periarteritis which often binds firmly together arteries, veins and nerves in one solid cord. Histological studies in some cases show also distinct interstitial and parenchymatous neuritis and atrophic changes in voluntary muscles, which probably explains some of the trophic manifestations. The evident attempted establishment of a compensatory circulation both by the vascularization of the occluding thrombus and by utilization of natural anastomotic routes, shows the tendency to self-cure, or at least to maintain a sufficient circulation.

These pathologic processes are of such a character as to suggest their being caused by some specific type of microörganism or toxin. No one, however, has succeeded in demonstrating the offending agent bacteriologically or morphologically.

Willy Meyer and Bernhard, as a result of their extensive work in testing out the sugar tolerance in their large group of cases suffering from thrombo-angiitis obliterans, contend that it is not caused by a specific organism but that the occluding thrombosis is caused primarily by a stasis. This is brought about by the presence of a hyperglycemia and increase in the red blood-corpuscles and blood-platelets which produces a marked increase in the viscosity of the blood, which is in turn conducive to the production of variable degrees of stasis in the pendent vessels. The sugar concentration in the blood approached closely that found in diabetes. This led them to believe that the occluding thrombi were primarily and originally a conglomeration of red blood-corpuscles and platelets which later became organized. It is interesting to note that they were successful in relieving the distressing symptoms in many cases by reducing the viscosity.

Symptoms and Diagnosis.—The symptoms are due:

1. To the gradual shutting off or diminution of the arterial blood supply to the muscles by the occluding thrombus, this being most marked at times when a maximum blood supply is required, as in walking or exercising the extremities involved.

2. To the almost complete occlusion of important blood supply causing extreme interference with local function, pain, trophic changes and gangrene.

The history of the advent and development of symptoms is very much the same in most cases, the only difference being the length of time before the symptoms, depending on the completeness of the occlusion, are sufficiently marked to attract attention. The first sign noticed by the patient, and usually attributed to rheumatism or flat-foot, is pain in the toe, foot or calf of one leg while walking or standing. Sometimes at the onset the conspicuous symptom is a cramplike feeling in the calf, hip or lumbar region while exercising. I have seen a small group of cases where all the early symptoms were referable to the back and hip, suggestive of lumbago or sciatica. This pain is relieved when he sits down and holds up his leg, but recurs again on an attempt to walk a distance or otherwise exercise. This most characteristic symptom is present in most cases and is commonly spoken of as "intermittent claudication," which term was first used to describe the intermittent lameness in horses due to venous thrombosis from other causes.

After a variable length of time these patients are forced to take frequent rests while walking and one can judge the progress of the arterial occlusion in any one case by the distance he can walk without resting. I have seen patients, whom the pain would induce to stop, examine their affected limb and even take off their shoe and try to dispel the pain or numbness by rubbing. Others who do not at first suffer from "intermittent claudication" complain that their feet become cold and numb or burn when exposed to dampness or cold weather. Another common symptom is the feeling that they have stockings on, when walking barefoot. There are some abortive cases where, after the common symptoms have been in evidence for variable lengths of time, they disappear and are never in evidence again. This may be explained by the establishment of a sufficient compensatory circulation either by the natural collaterals or the ample canalization of the organized thrombus.

If the condition exists in the upper extremity, the progress of symptoms is usually much slower and less disturbing to the patient, because it is much easier to favor the hands from exposure to cold and dampness than it is the lower extremities. Coincident with the first signs of this disorder, the affected limb when held in the pendent position takes on a characteristic purplish blue color which is noticed in the toes and forepart of the foot and which afterwards extends up as far as the ankle and other surrounding parts. Conversely, on elevation of the affected part high above the horizontal, it becomes promptly blanched, and the ischemia may persist even in the horizontal plane. The color depends entirely on the extent of thrombotic occlusion or the success of the collateral circulation.

In most cases, after a lapse of some months or years, as the thrombosis develops more extensively, first the dorsalis pedis pulse is cut off, then the posterior tibial, and finally the anterior tibial. Evidences of trophic disturbance now make their appearance either after a minor traumatism or without any apparent cause. An abraded spot, a hemorrhagic bleb or a small patch of superficial dry gangrene, appears, usually on the great toe, under the nail or on the heel. This usually spreads up the foot and finally up the leg.

It is at this time that severe pain becomes a marked symptom, first only when walking and later excruciating even at night in bed. So severe is the pain that patients oftentimes beg for surgical interference, even amputation, hoping for relief. Such severe symptoms, however, may take years to develop, because of the establishment of a collateral circulation or of a change of occupation requiring little walking or standing. Sooner or later, minor trophic manifestations turn into ulcers, and ulcers give way to gangrene, accompanied by excruciating pain, which is much worse when the foot is elevated than when on the floor. This is because of the greater local anemia incident to the elevated position. That angle at which the erythromelia disappears and the ischemia begins to appear has been recorded for each individual case for the purpose of noting the rapidity of the occluding process, and has been called the "angle of circulatory sufficiency." This serves as a method of checking up the extent of improvement in using the various methods of treatment.

It should be noted that one sometimes meets cases with this condition which have for a long time suffered symptoms consistent with occluded main arteries, but still have good pulsation in the dorsalis pedis and tibial arteries.

To summarize, the following are the important features upon which a diagnosis can be made:

1. A history of intermittent claudication in the foot or calf of one in middle life of the male sex and Jewish race.
2. Development of vasomotor unbalance and circulatory disturbance dependent upon the closure of peripheral vessels.
3. History of phlebitis, past or present, single or migrating, in any superficial vessel.
4. Presence of certain objective signs: ischemia upon elevation of the limb, erythromelia in the pendent position.
5. Local pain (constant or spasmodic).
6. Absence of peripheral pulse.
7. Appearance of trophic disturbance, such as ulcers.
8. Development of small areas of dry gangrene.

Prognosis.—One must be very guarded in making a prognosis in these cases. Practically all have a tendency to progressive occlusive thrombosis, finally ending in amputation and re-amputation, but sometimes very slowly. There are also those cases described as "abortive cases." In general, if it is possible to make an early diagnosis, especially in the stage when the symptoms are obscure and indefinite, and to direct and organize the future life of each patient in such manner that he can protect the affected parts from exposure and injury, to regulate his diet as to carbohydrates, and to change his occupation to one of less walking and standing, a longer and more useful life can be promised.

Treatment.—The purpose of all forms of treatment is the relief of pain, the restoration of function, or both. The general plan of the various methods that have been employed has been to increase the local capillary hyperemia, to encourage the formation of a collateral circulation that will be sufficient for the needs of all the muscles involved in the local functions, and to reduce the viscosity of the blood.

The final outcome in most cases in spite of all treatment, until the specific cause and cure are found, is and will be amputation, and to be successful must be high amputation. But since the severe symptoms may take years to develop, or, if a sufficient collateral circulation is established around the thrombosed vessels may never reach a stage requiring such radical procedure, so for the interim between the first beginning incapacity and the stages demanding amputation, many forms of treatment have been suggested and tried.

Drugs.—The drugs most commonly tried are potassium iodid, mercury, nitroglycerin, nitrites, benzyl-benzoate and all the other vasodilators, fibrolysin and thiosinamin. When the condition is associated with arteriosclerotic hypertension relief is seen for a time from the iodids and nitrites. In some of the early cases where the thrombosis is not extensive there is often relief of pain and the intermittent claudication on giving frequent small doses of benzyl-benzoate, but the result, if any, is not lasting.

Morphin and its derivatives will stop the pain, but on account of the great length of time that it has to be administered, and the ever-increasing doses that are necessary, is inadvisable. Almost equally good results can be had by using some of the newer salicylate products, such as aspirin, or atophan in combination with some hypnotic at night.

Locally, for small ulcers or broken down patches of gangrene, the usual surgical measures hold good, using mildly antiseptic powders for dry dressings and balsam of peru for the broken down areas. I might emphasize that it is well worth the effort and time spent in getting these little areas of gangrene healed up, because if neglected or infected they spread very rapidly.

Heat and Elevation of Member.—For the relief of severe pain and for healing up small gangrenous ulcers, elevation of the leg or arm and exposure to various forms of dry heat have their merits. The greatest measure of benefit is obtained by using a quartz lamp, or a high-powered electric lamp with a reflector, or an electric light bath. Where these are out of reach the even heat of an electric heating pad will serve fairly well.

The wound should be dressed often and be kept perfectly clean and dry. It must be borne in mind that this treatment may have to be carried out for weeks, but may delay an amputation and is therefore worth while. The use of any form of moist dressing is to be discouraged because it leads to maceration and spread of the destructive process. On the other hand, if there is no gangrene or ulceration, soaking the parts involved in hot water several times a day will relieve the pain and aid in maintaining function by producing a hyperemia.

Electrotherapy.—Dr. N. H. Ludington of New Haven has had occasion to use the d'Arsonval current in a number of cases, particularly in the stages of severe pain and beginning gangrene, and reports material improvement where this method has been used for a period of months: relief of pain, healing of gangrenous finger tips, and bettering of the circulation in the lower extremities to such an extent as to make it possible to amputate much more peripherally in cases finally requiring it. His technic is as follows:

> "Seated on the autocondensation pad with the other electrode applied to the immediate neighborhood of the lesion and distally, if possible, the patient is given the smallest milliamperage that can be achieved with the apparatus available. The current is allowed to flow for ten seconds and interrupted for five seconds. This is continued for twenty minutes to half an hour daily, or as frequently as every four hours in urgent cases. Where practicable a suitable electrode may be substituted for the condensation pad and applied high up on the affected limb, observing the rules as to size of electrode that obtain in diathermy. It has seemed that the best results have been obtained by the frequent interruption of the current in either form of treatment. The greatest care is essential to avoid coagulation."

Bier Suction Cylinders.—This method of producing a local hyperemia was suggested and tried out especially for the relief of pain. The hyperemia so produced was so sudden and transient that often it intensified the pain and was therefore given up as a general therapeutic measure.

Femoral Vein Ligation and Arteriovenous Anastomosis.—About 1910, Buerger conceived the idea of doing arteriovenous anastomosis in advanced cases, thus diverting the blood from the closed arteries to the open veins. His method was to do an anastomosis between the femoral artery and femoral vein, expecting that the venous return would be reestablished through the saphenous veins. The results were far from gratifying. Those whose deep veins were not thrombosed did well for a while but later came to amputation.

Lillienthal, in 1915, tried multiple ligation of the large superficial veins to produce a permanent hyperemia but without lasting success.

Nathaniel Ginsberg, in 1916, tried femoral vein ligation to produce circulatory stasis in the affected limb, in combination with repeated intravenous injections of 2½ per cent sodium citrate solution to reduce the blood viscosity and reported a fair measure of success in some cases. This benefit was probably more from the latter procedure than from the vein ligation.

Intravenous and Subcutaneous Injections.—Intravenous and subcutaneous injections of solutions for the purpose of lessening blood viscosity (sodium citrate or Ringer's solution) have been employed. In 1915, Mayesima demonstrated that there was a constant high viscosity of the blood in all types of gangrene. Acting on this knowledge Koga and others used Ringer's solution subcutaneously over long periods of time, and reported many cases of cure or improvement.

During 1917-1918 Meyer adopted a method of producing quickly the highest possible degree of hydremia in order to counteract the dehydration of the tissues involved (produced presumably by the increased viscosity) which was as follows:

"Intensive duodenal flushing with Ringer's solution or sodium bicarbonate solution using Einhorn's duodenal tube and about 8 to 10 quarts of the solution in twenty-four hours, combined with frequent hypodermoclysis." He reported the following results: "Pain not controlled by any other method, gradually improved, patient able to sleep with feet in bed and often a return of pulsation in artery." If amputation became necessary he could amputate more peripherally if the system was thoroughly permeated with fluid.

The latest contribution to this therapy comes from William Steele of Philadelphia (1921), who reported most brilliant results, namely:

1. Relief of pain after the second injection.
2. The checking of the gangrene, followed by spontaneous amputation of the dead tissue.
3. Healing of indolent painful ulcers.
4. Slow but sure establishment of a collateral circulation, as shown by improved color, warmth of foot, the filling out of the subcutaneous veins, and a strengthening of any existing pulse.
5. Finally a return to the pursuit of a gainful occupation.

Steele's technic is as follows: During the first month the patient is kept in bed with the legs constantly under a hot-air electric light bath at 110° F.; 250 c.c. of 2 per cent sodium citrate solution is given intravenously every second day. The second month the interval of injection is lengthened to every third or fourth day; daily leg massage is given and the patient is put in a wheel chair with the feet hanging down a short time each day, or some walking is allowed. The intervals of injection are now gradually lengthened until at the end of a year the patient gets one every two weeks. As the pain subsides and evidence of a functional collateral circulation appears, increased walking is permitted. The injections are given in the veins of the arm. The length of treatment is regulated by the results obtained in establishing a functional collateral circulation. Potassium iodid, 10 drops three times daily, is given during the whole course of treatment.

I have tried out this method in 7 cases, but of necessity was forced to shorten the duration of treatment. While there was decided improvement in some of the cases, the results were not as encouraging as those described. But certainly it is the only thorough method of medical treatment so far described and is well worth trying in every case, even if amputation is to be the eventual outcome.

Leriche's Periarterial Sympathectomy.—This operation was described by Réné Leriche of Lyons, France, in 1913, and applied to the surgical removal of the sympathetic nerve fibers in the wall of an artery. He suggested performing this operation in vasomotor and trophic disturbances of the region supplied by an artery. Leriche presented evidence that, after completely decorticating a vessel for a distance of from 8 to 10 cm., and thus destroying the vasoconstrictive element of the sympathetic fibers at this site, he found a marked paralytic vasodilatation peripheral to the point of operation and also an elevation of surface temperature which persisted for many days from 2 to 3 degrees over those parts distal to the operation. This reaction of vasodilatation with hyperthermia has also been noted after sympathectomy by many other observers, but not with such uniform success.

The purpose of this operation in thrombo-angiitis obliterans is quite evident. If it is possible to increase the blood supply and surface temperature of the peripheral parts by performing a decortication of the artery supplying that part, we would have at our command a method of promptly relieving most of the predominating and distressing symptoms. This operation has been of great help in some cases which did not respond to other forms of treatment and were considered hopeless.

The objections to this operation, as reported by a large number of surgeons, are as follows:

1. The results have not been uniformly successful, and have not agreed entirely with those reported by Leriche.
2. The "perivascular sympathectomy" of Leriche does not result experimentally in the dog, rabbit, and cat, in the physiological changes in the extremities that are described by him as resulting in clinical cases.
3. Recent discoveries made in the study of the anatomy and physiology of the sympathetic system make it difficult to explain the clinical phenomena as reported by Leriche.

Technic (*from the W. S. Halsted translation*).—"Uncover the artery by the classic procedure, open the cellular sheath with the bistoury, separate the artery from 8 to 10 cm., grasp the inner sheath directly on the vessel wall, incise it, pull one of the lips thus made with the forceps, free it either with grooved probe or with a bistoury, completely stripping the artery, to decorticate a fold of all the cellular tissue that adheres to it. . . ."

DISEASES OF THE LOCOMOTOR SYSTEM

CHAPTER XXI

DISEASES OF THE MUSCLES

CHARLES LYMAN GREENE

REVISED BY GEORGE BLUMER

VARIOUS FORMS OF MYOSITIS

Acute Polymyositis (*Dermatomyositis—Pseudotrichinosis*).—First described by C. Wagner as dermatomyositis in 1886, this disease presents a peculiar combination of almost universal progressive interstitial myositis of high grade, with a considerable fever (104° F.), severe pain, acute tenderness, marked and ultimately complete disability, edema and redness of the overlying skin and involvement of the subcutaneous tissues. The muscles show more or less marked induration.

The onset is usually gradual, with the usual symptoms of an acute infection, but lacking chill and high fever.

This stage is succeeded by severe cramplike pains and marked tenderness in the affected muscles.

The disease usually attacks the proximal muscles, first of the upper, later of the lower extremities, and extends distally.

The tongue, muscles of deglutition, ocular muscles, diaphragm, and accessory muscles of respiration usually escape.

Cases have been reported by a number of clinicians, and the disease seems to represent a distinct clinical entity. Mild cases occur in which but few or even one muscle alone may be affected.

The spleen is usually enlarged, and albumin may or may not be found in the urine.

The specific cause is unknown, but its infectious nature seems to be clearly indicated.

Von Niedner isolated a streptococcus in one patient.

It is unlikely to be confounded with any ailments save polyneuritis and trichinosis. From the former it is easily distinguished by the seat and distribution of its pain and tenderness. From the latter, by its appearance as an isolated case, the absence of eosinophilia and of parasites, and the rarity of early involvement of the eyelids, jaws, or larynx.

Examination of the blood, the stools, and of a bit of the muscle itself will also serve in any case of peculiar difficulty, and such a procedure must often be essential to an unequivocal diagnosis.

The prognosis is grave in severe cases, good in the case of limited lesions. The disease seldom subsides within a period of less than a month or six weeks, may endure for months or become chronic and last for one or two years. Occasionally the disease recurs years after the original attack.

Cases in which the symptoms are more trivial and evanescent are reported but seem atypical and, in many respects, dubious.

Treatment.—Lenhartz and Stinzing advise the hot, dry packs, support, elevation, and relative immobilization of the affected members. The use of cotton packing and light bandaging to secure warmth and, if possible, to exert gentle continuous pressure is desirable.

Aside from these means relief of any persisting pain must be sought through the use of aspirin, salipyrin, phenacetin, antipyrin, or salicylates and morphin may be employed if other means fail. In persistent or chronic cases massage is valuable, as are faradization and galvanization of the muscles affected.[1] Gymnastic exercises are of value, but care should be taken to increase the exercise very gradually.

A weakened heart may require appropriate stimulation, and a generous diet as soon as the fever will permit. Rest in bed is essential in the severer cases and at the onset of the milder ones.

Syphilis of the Muscles.—Syphilis of the muscles occurs in three forms:

1. The diffuse myositis of secondary syphilis associated with nocturnal pain, some disturbance of function, local tenderness but no obvious changes in size or shape. This form of muscle syphilis subsides under the general treatment for the secondary stage but, in addition, stimulating applications over the affected muscles are of value and baking or the Turkish bath are often of service.
2. Diffuse gummatous infiltration of muscles. This most commonly affects the muscles of the upper arm or thigh but may occur elsewhere. Single muscles are often involved and show a painless, or almost painless, increase in size with disability from shortening. The lesion occurs in the early tertiary stage. In these cases general treatment is needed and may be applied in the form of mercurial inunctions over the affected muscle with massage.
3. Localized gummatous muscle tumors in late hereditary syphilis or in the late tertiary stage of acquired syphilis. The tumors occur most commonly in the sternomastoid, quadriceps femoris, calf muscles and

---

[1] In one case of dermatomyositis, collargolum given in suppository seemed to have the most remarkable effect, the patient recovering.—Editor.

upper arm muscles and the gummata are associated with surrounding diffuse myositis. Mixed treatment is indicated in these patients.

Primary Acute Suppurative Myositis.—Acute suppurative myositis is a rare acute infectious disease characterized by inflammation of a single muscle or of several muscles, terminating in diffuse purulent infiltration, localized single abscesses, or multiple purulent foci.

The disease affects but one muscle in about one-half the cases, and seldom involves more than two.

First described in 1869 by Foucault, it has been especially studied by Suard, Nicaise, Walther, Brunon, Scriba, Lorenz, and Steiner, while certain Japanese investigators, notably Miyake, have added much to our knowledge of the disease and reported 250 cases.

A large literature is now available bearing upon the etiology of the disease, but so far no specific germ is proved, and the Staphylococcus pyogenes aureus seems to be most constant in the lesions. Neither has any definite portal of entry been discovered, and it would seem that the sources of infection may be varied, and the muscle injury and overstrain are the chief predisposing factors.

It affects the male more frequently than the female, is seen chiefly in young adults and children, and is encountered usually among the laboring classes. The etiologic factors are thoroughly discussed in Steiner's admirable article.

The clinical picture is that of an acute suppurative process. The onset is usually sudden with chill or chilliness, fever and sweating, general prostration and bone joint and muscle pains. A swelling soon becomes manifest in the muscles, which are extremely tender and painful.

In the long muscles the induration is spindle-shaped, in the broad muscles hemispherical.

The signs of suppuration soon become manifest in most cases, and the treatment is essentially surgical.

The affected muscles are extremely painful and their tonic contraction involves corresponding changes of a fixed character in the position of the members or portion of the trunk affected.

Prompt recognition of the nature of the ailment and appropriate and judicious surgical treatment are usually effective and the actual mortality is low.

Atrophy of the muscles or extensive replacement of muscle by scar tissue may cause more or less marked deformity, but usually the patient's recovery is uneventful.

Treatment.—Antiphlogistic local measures such as the use of cold applications are desirable because suppuration may not take place. Where there is definite proof of pus formation prompt evacuation and surgical treatment is demanded. During convalescence the tendency of these muscles to permanent contraction must be borne in mind and massage and passive motion must be employed.

Secondary Suppurative Myositis.—It is unnecessary to discuss, under a separate heading, the manifold instances of this complication of the acute infectious diseases from glanders through the long list embracing erysipelas, typhoid, malignant endocarditis, etc.

Tuberculous Myositis.—This is one of the rarest forms of tuberculosis and occurs in one of two forms: (1) localized tuberculosis of muscle tissue in the immediate neighborhood of tuberculous bones or joints; and (2) multiple muscle tumors or abscesses connected with the generalization of a tuberculous process. The muscular nodules in the latter type of the disease develop slowly and painlessly and break down at a late period. They generally occur in the muscles of the forearm, thigh or upper arm.

Treatment.—Aside from treating the patient for the general tuberculosis along the usual lines, incision of the broken down nodules is indicated. In some patients the local use of the X-ray has been beneficial.

Myositis Fibrosa.—This condition is almost invariably a distinctly secondary process associated with simple or septic chronic inflammatory changes in the muscles or the neighboring joints. It may play a part in primary or spinal muscular atrophies or even follow extensive chronic eczema and ulcer.

Treatment.—The treatment is largely limited to massage, passive movements, and orthopedic resources.

Myositis Ossificans.—This extraordinary disease is a clinical curiosity and its victims among the poorer classes are likely to fall into the hands of the side-show and dime-museum proprietors.

The extraordinary pathologic change involves not alone the muscle fibers but the interstitial connective tissue, the aponeuroses, and the tendons.

Injury, chronic disease of the joints, excessive muscular strain and the like are usually the antecedent factors. There are three types of the disease the recognition of which is important from the therapeutic standpoint:

1. The type which follows a single violent injury to muscle with tearing loose of periosteum. This occurs in football players, horseshoers or hostlers and in posterior dislocations of the elbow joint.
2. The type which follows repeated traumata of the same muscle or group of muscles, that is, "rider's bone," "exercise bone."
3. A spontaneous progressive polymyositis ossificans which usually first develops in youth. In such cases the muscles of the back and neck are likely to be first involved.

The disease passes through stages representing (*a*) infiltration, (*b*) fibrous deposits, (*c*) bone formation.

Treatment.—In the type of the disease which follows a single severe trauma the bony mass may interfere with the function of the affected muscle, may involve a nerve causing severe and continued pain, or may interfere with the blood supply by pressure on or inclusion of a vessel.

In such circumstances operative interference is demanded, excision of the mass is indicated where muscle function is affected, freeing of the involved nerve is required where there is nerve pressure, and the removal of pressure on affected vessels is indicated where these are involved. *The operation must not be performed before the X-ray has determined that the process is at a standstill* or recurrence may occur.

MUSCULAR RHEUMATISM

(*Myalgia*)

There is serious question whether the condition commonly spoken of as muscular rheumatism or myalgia constitutes a disease entity. The histological examinations of muscle tissue from affected individuals have been few in number and have usually been negative. We are compelled, therefore, to consider the treatment from the viewpoint of clinical observation rather than to base it upon a firm foundation of pathological facts. Clinical and bacteriological observation points to certain factors as being of etiological importance. These are in brief:

1. *Focal infection,* especially in the teeth, tonsils and sinuses.
2. *Trauma,* that is, continued overuse of particular muscles or groups of muscles either as a result of occupational bad posture or overstrain from congenital or acquired deformities, such as flat feet, partial paralysis, spinal curvature, etc.
3. *Chilling,* especially after overheating accompanying violent use of single muscles or groups of muscles.
4. *Toxins,* either exogenous (lead, arsenic or alcohol), bacterial, or metabolic (gout, kidney disease).
5. *Circulatory disturbances,* either arterial anemia or venous hyperemia.
6. *True rheumatism,* the myalgia either accompanying the arthritis or occurring alone in an individual who has suffered from rheumatic arthritis.

The clinical manifestations of the disease take the form of pain and disability in certain muscles or groups of muscles, notably those of the neck (stiff neck), the trunk (pleurodynia) and the back (lumbago). The onset is frequently startlingly sudden, the condition is afebrile or nearly so except in the true rheumatic cases and the course is very variable, some cases clearing up spontaneously in a few days, others persisting for months. Physical examination frequently shows, besides limitation of motion from pain, a local increase in heat, localized points of tenderness and palpable nodules in the affected muscles which are probably areas of localized spasm.

It is clear from the above considerations that the *prevention* of the disease must depend on the etiology. The main principles of prevention may be summed up as follows:

1. The removal of all detectable local foci of infection.
2. The avoidance of chilling, particularly when overheated.
3. The correction of deformities which result in faulty position with excessive strain on particular muscles or groups of muscles.
4. The removal of occupational strain.
5. The removal of sources of intoxication either exogenous or metabolic.

Treatment.—The treatment of the condition is likewise to some extent dependent on its etiology and should include the following:

Complete rest, demanded during the acute stages of the process. This may mean rest in bed where large muscles like the lumbar muscles are involved or local rest induced by strapping or other mechanical means if the location of the lesion renders this feasible.

Warmth in the form of the hot-water bag, the electric pad, the hot-air douche, diathermy, the domestic flat iron or the Pacquelin cautery, the therapy of next greatest importance.

Thorough evacuation of the bowels, demanded at the onset of the disease.

Free water drinking, to aid the elimination of toxins.

An appropriate diet, if metabolic disturbances are found.

Medication in the form of salicylates or atophan. Where the pain is intense morphia may be necessary.

There are differences of opinion as to the dosage of salicylates that is demanded. Some writers, F. Schultze, for example, state that large doses are unnecessary. Others, such as Quincke, say that too small doses are usually given and that from 4 to 8 gm. daily (60 to 120 gr.) should be given.

In protracted cases, after the intense pain has subsided, other methods are applicable, such as massage, faradism, the local injection of normal saline solution, or acupuncture.

The massage should take the form of gentle centripetal stroking until the patient has become accustomed to this. Then rubbing with the fingers transverse to the course of the muscle should be substituted, at first superficial but gradually becoming deeper. This may provoke severe pain but is usually followed by relief. Daily sessions of from ten to fifteen minutes should suffice. If a severe reaction follows the treatment it should be discontinued for a day or two (Lange and Everbusch).

The technic of acupuncture is as follows: The skin over the affected muscle is cleansed and sterilized with iodin and alcohol. Ordinary bonnet pins or darning needles or a hypodermic needle may be used. One or more needles are sterilized and inserted into the muscles at the painful points at right angles to the body surface. The depth of insertion depends on the site of pain, usually from three-quarters of an inch to an inch suffices. The needles may be left in from five to ten minutes. If a steel needle is used it generally shows signs of oxidation on removal. The procedure may be repeated if necessary. Immediate and striking relief frequently follows.

MUSCULAR CRAMPS

These affect chiefly the calves of the legs, the feet, and less frequently the other muscles of the lower extremities. They come on suddenly, more often at night, and are extremely painful and troublesome. They may be only momentary in duration, but often last for from fifteen to thirty minutes, and they occur almost wholly in persons suffering from overexertion or some form of acute or chronic intoxication.

One encounters them in connection with gout, chronic nephritis, diabetes, alcohol, lead, and arsenic poisoning, and in connection with overuse of the muscles associated with extreme fatigue. Cholera, typhus, and even acute diarrheas or dysenteries are well-recognized causes.

Patients suffering from the results of thrombosis of the femoral vein or calf veins, and patients who have survived a severe attack of trichinosis not infrequently have them. They are common in connection with obliterating processes in the arteries of the lower extremities.

Treatment.—Friction, kneading, and hot applications give most relief.

The sudden application of a cold substance to the sole of the foot is sometimes effective when other measures fail.

The cause should be sought and receive appropriate treatment. If due to overuse of the muscles a supporting bandage is often of value.

LOCALIZED RUPTURE OF MUSCLES

Aside from the complete or partial rupture of muscles which may occur from overaction, rupture of the plantaris or of the long head of the biceps for example, slight local ruptures of muscle occur in connection with certain infectious diseases, notably typhoid fever and influenza. These lesions usually pass unnoticed by the patient and physician but at times they give rise to hematomata which are visible and palpable and may undergo secondary suppuration. Such lesions nearly always occur in the rectus abdominis.

Treatment.—The treatment is generally entirely expectant as the patients are usually in bed at the time rupture occurs. Pain is seldom severe, indeed it is usually absent, and when it occurs is controllable by the milder sedatives such as bromids. When secondary infection occurs a local abscess forms which must be evacuated and drained and then treated on general surgical principles.

REFERENCES

Abram. Suppurative Myositis, Brit. Med. Journ., ii, 1341, 1904.
Batten. Myositis, Allbutt's System of Medicine, vii, 3, 1911.
——— Myositis Fibrosa, Tr. Clin. Soc., London, xxxvii, 12, 1903-1904.
Bechtold. Uber zeitweiser gehaüftes Vorkommen von Endocard. bei Muskelrheumatismus, München. med. Wchnschr., xlv, 2189, 1906.
Burley. Subacute Polymyositis, Journ. Am. Med. Ass., l, 177, 1908.
Clowe. Acute Primary Polymyositis, Report of a Case, N. Y. Med. Journ., cii, 1177, 1910.
Edsall. Metabolism in Relation to Muscular Disorders, Bull. Manila Med. Soc., iii, 17, 1907.
Elliott. Myositis Ossificans Progressiva, Journ. Am. Med. Ass., lvii, 873, Sept., 1911.
Foucault. Myositis, Bull. Soc. anat. de Paris, 506, 1869.
Forchheimer. Dermatomyositis, Tr. Ass. Am. Physicians, xviii, 184.
Godlee. Myositis Ossificans Traumatica, Proc. Royal Soc. Med., Surgical Section, iv, 144, London, 1910-1911.
——— Lancet, lxxix, 1911.
Hepp. Uber Pseudotrichinose, etc., Berl. klin. Wchnschr., xvii, xviii, 1887.
Heyerdahl, C. Rheumatic Symptoms in Muscles and Fibrous Tissue and Massage, Ugesk f. Læger, Kbenh., lxxiii, 16, 545, April 20.
Kader. Myositis, Mittheil. a. d. Grenzgeb. d. Med. u. Chir., 11, 617, 1897.
Kaufmann. Myositis, Spezielle pathologische Anat., 1186, 1909.
Klose. Uber eine selene Lokalisation der Myositis Ossificans Traumatica, Ztschr. f. orthop. Chir., Stuttg., xxviii, 385-391, 1911.
Knowles. Note on Case of Nodular Fibromyositis, Lancet, i, 734, 1911.
Ledderhose. Uber akute Myositis, Strassb. med. Ztg., viii, 127-129, 1911.
Lenhartz. Handbuch der Gesamten Therapie, Penzoldt und Stinzing, v, 107, 118, 119.

Lenhartz and Stinzing. Ibid., v, 436, 1911.

Leube. Beiträge zur Pathologic des Muskelrheumatismus, Deutsche med. Wchnschr., 1, 1894.

Makins. Traumatic Myositis Ossif., Proc., Royal Soc. Med., Surg. Sec., 134-142, London, 1910-1911.

Marcus. Fall vom Myositis ossificans traumatica, Aerztl. Sachoerst. Ztg., xvi, 468, Berlin, 1910.

Miyake. Suppurative Myositis, Mittheil a. d. Grenzgeb. d. Med. u. Chir., xiii, 153, 1904.

Nicaise. Suppurative Myositis, Revue mens. méd. et chir., i, 51, 1877.

Oppenheim. Lehrbuch der Nervenkrank., 5th ed., i, 1908.

Parès. Myosite Sclereuse du Quadriceps Traitée par la Mécanothérapie, Montpel. Méd., xxxiii, 110-113, 1911.

Pouken. Myositis ossif. traum., Hosp.-Tid. Kbenh., iv, 681-705, 1911.

Praag. Myositis Ossif. Circumscripta, Med. Weekbl. Amst., xviii, 177-191, 1911-1912.

Salinin. Primary Tuber. Myositis, Sibirsk Vrach. Gaz., Irkutsk, iv, 230, 1911.

Salzberger. Myositis Ossif., Symptomatic Myotomic Disturbances with Inflammation in Muscles, Berl. klin. Wchnschr., xlvii, 49, 2225-2276.

Schmidt. Das Problem des Muskelrheumatismus, Med. Klin., 19, 1910.

Steiner. Dermatomyositis, Rare Muscle Anomaly, Journ. Exper. Med., vi, 407, 1903.

——— Myositis, Osler's Mod. Med., vi, 575-594.

Suard. Myosite aiguë suppuré, Dissertation Inaugurale, Bordeaux, 1887.

Tubby, A. H. Torticollis or Wry-neck, Practitioner, lxxxviii, 1, 55, Jan., 1912.

Unverricht. Polymyositis acuta progressiva, Ztschr. f. klin. Med., xii.

Waetzold. Beitrag zur Lehre der Polymyositis, Ibid., xxii.

Wagner. Dermatomyositis, Deutsche Arch. f. klin. Med., xl. 241, 1886-1887.

Zeller. Myositis, Beitr. z. klin. Chir., xxxix, 633, 1903.

CHAPTER XXII

DISEASES OF THE BONES

HENRY JACKSON, JR.

OSTEITIS DEFORMANS

Synonym.—Paget's disease.

Definition.—Osteitis deformans is a chronic, progressive disease of the skeleton, beginning usually in middle life or later, and leading to a painful thickening and bowing of the long bones, enlargement of the head, dorsocervical kyphosis and deformity of the thorax.

Etiology.—It is considered by many a rare disease. Locke, however, regards it as the commonest of the chronic bone diseases, if syphilis be excepted, and Da Costa believes that "the more carefully we are on the lookout for cases, the more of them we will discover."

The cause of the disease is unknown. Syphilis, gout, trauma, bacterial infection, heredity, malignant disease and finally endocrine disturbance and arteriosclerosis have all been suggested as possible etiological factors. Generalized arteriosclerosis is an almost constant accompaniment and the disease seems to be hereditary in not a few instances. It is an affection essentially of middle and late life and, while cases under twenty have been reported, it is a question whether these were instances of true Paget's disease.

Pathology.—There is general thickening and bowing of the long bones. The skull may be enormously thickened. The sella turcica may be flattened and shallow. Rarefaction and excessive new bone formation, poor in lime salts, are simultaneous processes. There is retention by the body of calcium, magnesium and phosphorus.

Symptoms.—The onset is insidious. Superficial pain over the affected bones, enlargement of the head and stiffness on motion are early symptoms. The tibiæ and tables of the skull are among the first bones to be attacked. Failing vision, cramps in the lower legs and muscular weakness are not uncommon. There may be pigmentation over the involved areas. Gradually the head enlarges, the limbs bow, the knees and hips flex and the body is shortened by the anterior curvature of the spine so that the costal border of the thorax may come to rest on the iliac crests. The "anthropoid ape" appearance is striking and characteristic. X-ray is an important diagnostic aid particularly early in the disease when perhaps but one bone is involved. Fractures caused by minimal injury are not uncommon. Arteriosclerosis of an extraordinary degree is a very constant finding.

The disease is chronic and generally progressive, though it may rarely come to a standstill. Death is usually caused by cardiorenal complications or malignant disease. More rarely pulmonary infections close the scene.

Treatment.—The pain may be relieved, when not too severe, by salicylates. Recourse to opiates should be had only in extreme cases in view of the chronicity of the disease. Local heat may temporarily alleviate the distress and trephining of the long bones is said by Da Costa to be of some help in certain cases. Orthopedic measures are generally useless, though some authors advise their institution. Surgical intervention with excision of the diseased bones has been tried in those cases where the involvement seemed very limited but the results have not been very satisfactory and in Romer's case the disease reappeared in other bones.

A high protein, low carbohydrate diet has been advised by Fairbank.

The general health should be improved as much as possible by rest, liberal diet and tonics. X-ray treatment has been advocated and internal gland therapy is at present advised by some, but it is doubtful if such measures will prove to be of any real value.

It is likely that in spite of all treatment the disease will progress and if the general health can be kept up and the pain alleviated as much as can be expected has been accomplished.

SECONDARY HYPERTROPHIC OSTEOARTHROPATHY

Definition.—Secondary hypertrophic osteoarthropathy is a condition, generally if not always, secondary to some other disease and characterized by symmetrical clubbing of the fingers and toes. There is, especially in the later stages, an associated thickening of the long bones of the lower arms and legs.

Etiology.—The disease is usually secondary to some pathological process in the lungs, heart or intestinal tract. Idiopathic cases have been reported, but more careful search in these instances might have disclosed a primary focus. The great majority of cases are due to pulmonary tuberculosis or bronchiectasis. Other common factors in the production of the disease are empyema, malignant tumors of the lung and mediastinum, valvular and congenital heart disease, biliary cirrhosis and chronic enteritis.

While the disease is associated with these varied processes, the actual cause of the pathological changes seems to be long-continued hyperemia in some cases and in others a toxemia.

Pathology.—There is thickening of the soft tissue of the affected parts, mossy proliferation of the distal phalanges, and in the more advanced cases a subperiosteal bony proliferation of the long bones. In the late stages there may be a low-grade synovitis leading to thickening of the joint capsule and erosion of the cartilage.

Symptoms.—In the early stages symmetrical clubbing of the fingers and toes occurs without any subjective symptoms. The onset is usually insidious, but the condition may develop in the course of a few weeks. The hands and feet seem large. The nails are markedly curved in both directions, thickened and often cyanotic. As the disease progresses the bones of the forearms and lower legs become affected. In very advanced cases the bones of the upper arm may also be involved. Pain sets in relatively early. It may be trifling, but is usually an ache deep in the bones and may be very severe, particularly during the exacerbations of the primary disease. In late stages the joints become involved, especially those of the wrists and ankles. Anemia may be present. Muscular atrophy is not uncommon and kyphosis has been described.

Treatment.—The treatment should be directed against the primary disease and in idiopathic cases every care should be exercised in looking for an underlying factor; for in its cure lies the only hope of alleviating the symptoms. The process may clear up rapidly following eradication of the primary focus. Counterirritation and local heat application serve to lessen the pain. Analgesics may be necessary.

OSTEOMALACIA

Definition.—Osteomalacia is an acquired, generally progressive disease most often seen in pregnant women, and characterized by general muscular weakness together with a widespread softening of preexisting bone leading to marked deformities and spontaneous fractures.

Etiology.—It is a rare disease in America and England, more common in Italy, Germany and France; almost unknown in Japan. It is a disease essentially of early adult life, occurs chiefly in women and certainly bears an unmistakable relation to pregnancy and lactation, each succeeding gestation usually advancing the progress of the disease.

The precise etiological factor is unknown but some evidence has been brought forth to show that disturbance of the glands of internal secretion, particularly the ovaries, parathyroids and pituitary gland, may play an important part.

Pathology.—The bones are abnormally soft and show on analysis a decrease of inorganic and a corresponding increase of organic constituents. The pelvis and spine are most seriously affected. Involvement of the skull is rare. There is constant body loss of calcium and phosphorus, though there may be periods when retention of these elements is seen.

Clinical Forms.—The vast majority of cases are associated with pregnancy or lactation. In the non-puerperal form the involvement of the pelvis is less severe and the onset is more gradual. Finally there is the "hunger osteomalacia" so commonly seen in the famine districts of Russia and Austria during and after the recent War. In this type pelvic deformity was rare, while pain in the lumbar spine and thorax was often severe and incapacitating. The relation of this form to true osteomalacia is uncertain.

Symptoms.—The disease is rarely recognized until it has reached an advanced stage. Dull or severe pain in the pelvis, back, hip or extremities is the earliest and most constant symptom. The muscles of the legs may be held rigidly in adduction. Muscular fatigue is prominent. Bony deformity is striking; the thorax is laterally compressed, the pelvis crumpled up; kyphosis and scoliosis result in a shortening of the body as a whole. The long bones, particularly those of the leg, are bowed. Multiple fractures are not uncommon. Muscle wasting may be extreme and ankylosis is not at all rare late in the disease. The X-ray shows an extraordinary rarefaction of the bony tissue.

Course and Progress.—The disease is usually chronic and progressive, but remissions do occur and spontaneous cures have been reported. Exceptions of this sort should be remembered in judging the effects of therapy.

Treatment.—General care of the health is most important. The patient should have absolute rest, adequate nursing and a full nutritious diet, rich in calcium and phosphorus (especially eggs, meat, beans, peas, other green vegetables and nuts). Mechanical supports may be necessary. If the patient has to be confined to bed for a long time, great care should be exercised to prevent bed-sores.

Fehling, in 1891, reported favorable results following castration and since his time this procedure has been followed in many cases, often with marked success. It is a question, however, as McCrudden first pointed out, whether the improvement following castration is not due to the prevention of further pregnancy; for numerous observers have shown that castration is without notable effects on the metabolism; while, on the other hand, it is certain that gestation exercises a markedly deleterious effect on the course of the disease. Furthermore, Edelmann has seen 5 cases of osteomalacia following hysterectomy and excision of the ovaries.

Cod-liver oil over long periods and Pil. phosphori, U. S. P. seem to help in senile cases. Hot packs may alleviate the pain. Cramer claimed that the milk of castrated goats produced favorable results. White, from his observations on animals, concludes that the starting point of treatment must be a diet rich in vitamins and containing a proper inorganic balance.

In view of the present-day enthusiasm for internal gland therapy, it is not surprising that this form of treatment has been warmly advocated. Bossi, in 1907, got good results from the administration of large doses of adrenalin subcutaneously. Scipiades found that the total extirpation of the thymus in young dogs produced the picture of human osteomalacia and concluded that thymic disturbance, particularly when associated with pregnancy, was the cause of the disease. Elfer and Kappel, in a careful and conservative series of experiments, conclude that there is but little value in internal gland therapy.

Recently Freund and Lockwood found that the administration of both thyroid and pituitrin increased the elimination of calcium, and were, therefore, worthless. In this they are supported by Falta and others. They were, however, able to attain favorable results, as far as chemical studies show, by the administration of parathyroid glands. But it should be remembered that the retention by the body of calcium and phosphorus is by no means synonymous with adequate utilization of these elements by the bony tissues and unless such utilization is made possible the therapy is valueless from the clinical point of view.

In general it may be said that the administration of internal gland extracts offers an interesting field for research and one that should be carefully pursued when opportunity offers; but there is no very clear evidence, as yet, that therapy of this sort is of real advantage.

Famine osteomalacia is more amenable, as would be expected, to rational treatment. Dalyell and Chick have found that recovery almost always followed the giving of cod-liver oil in sufficient quantities and they conclude from their large clinical experience that the curative value of the various oils used (other than cod-liver oil) was in direct proportion to their content of vitamin A. Hume and Nirenstein have come to the same conclusion and concur in recommending cod-liver oil as the curative agent.

REFERENCES

Osteitis Deformans (Paget's Disease)

Da Costa, J. C. Surg. Clin. North America, 47, 1921.
Fairbank. Proc. Roy. Soc. Med., London, Surg. Sec., xiv, 20.
Freire, L. Arch. de méd. d. enf., xxiv, 289, 1921.

Locke, E. A. Oxford Medicine, iv, 408.
Romer, F. Lancet, No. 1, 77, 1921.

SECONDARY HYPERTROPHIC OSTEOARTHROPATHY

Becher. Ztschr. f. klin. Med., lxxxiv, 491, 1917.
Hoffman. Deutsches Arch. f. klin. Med., cxxx, 201, 1919.
Locke, E. A. Oxford Medicine, iv, 431.

OSTEOMALACIA

Dalyell and Chick. Lancet, ii, 842, 1921.
Elfer and Kappel. Ztschr f. exper. Path. u. Therap., xxi, 104, 1920.
Freund and Lockwood. Ann. de méd., 1, 67, 1920.
Hume and Nirenstein. Lancet, ii, 849, 1921.
Locke, E. A. Oxford Medicine iv, 462.
Scipiades. Ztschr. f. Geburtsh. u. Gynäk., lxxxi, 156, 1919.

DISEASES OF THE KIDNEYS

CHAPTER XXIII

MECHANICAL AND CIRCULATORY LESIONS

FREDERICK FORCHHEIMER

REVISED BY GEORGE BLUMER

MOVABLE KIDNEY

(*Enteroptosis*)

Movable organs are not uncommon; Hector Mackenzie examined 2,801 females of all ages and found movable kidneys in 516.[1] In 1,607 males a movable kidney was found only 11 times. These statements establish the fact, already known, that enteroptosis is much more common in females than in males. The cause of this is easily understood by considering the different conditions which exist in the male and female that make for the support of the abdominal organs. The uterus and adnexa, originally in a horizontal position, now have the same supports for an upright position of the body. During the process of evolution the supports have not changed, so that, in the female, there is weakness of support for the pelvis and indirectly for the abdominal organs. The abdominal organs are held in place by the abdominal walls, and all those conditions, childbirth, tight lacing, etc., which weaken them may and do cause this condition. There is intra-abdominal support, as well; this is accounted for by the intra-abdominal fat; for the kidneys especially there is a thick layer of fat surrounding them. Intra-abdominal vascular tonus should also be considered; the more it is diminished the less support there is to the intra-abdominal organs. Indirectly this affects the kidney. It is thoroughly recognized that frequently two, or even more, organs are out of place at the same time when the external supports are normal. This can only be due to some intra-abdominal cause, and when the fat is normal the circulatory condition accounts for it. Anything acting mechanically, such as strain in lifting, traumatism, or increase in weight of the kidney, may be followed by dislocation of the kidney; literally the kidney is dislocated, but it is doubtful if this condition should be considered with that of enteroptosis. Certainly, for therapeutic purposes, this is not the case.

---

[1] It is perfectly normal to find a movable right kidney in most women and many thin men. A sharp distinction must be drawn between normally movable and pathologically movable kidneys.—Editor.

In treating these patients the physician should be careful how he communicates the fact that there is a movable or floating kidney. If, as seems to be the case, about 20 per cent of these patients do not know that the kidney is displaced (Mackenzie), it is well for the physician to study his patient before he makes the communication. Many a patient has been made an invalid by failure to follow this rule; neurasthenic, hypochondriacal, and sometimes psychotic. The treatment should be conducted along causal indications. It is not necessary to emphasize the fact that, when there is no weakness in the external abdominal supports, a bandage is only of symptomatic value. When there is this weakness a bandage should always be applied. The principal requirements for a good bandage are that it be supported by the most rigid base that can be found, in this disease the ribs and the pelvis; that it be sufficiently rigid itself to form a good support for the pad (or pads) that must be added to hold up the organ (or organs); and, finally, that it move not too much or too little—too much in that insufficient support is given; too little in that respiratory movements and the necessary movements of the body be interfered with. The designing, fitting, and application of the bandage should be under the supervision of a physician who is prepared to undertake such cases. In place of a bandage, a special supporting corset, which is less cumbersome, may be used. In the case of any kind of support it is very important that the patient be instructed that it must be put on in the recumbent position with the hips elevated. A support is worse than useless if applied with the patient standing.

In the case of tight lacing the cause should be removed; sometimes this is deferred too long and then nothing can be done for the condition except surgical treatment. When the diagnosis is made early enough, and the results of the condition are explained to the patient, many get well without further treatment.

Those cases in which intra-abdominal support is at fault do well enough when the cause can be removed. Not infrequently in these cases we find a combination of causes, flaccid abdominal walls and lack of fat intra-abdominally, the first to be treated by the bandage, the second by diet and rest. The object of dieting should be the making of fat; for this purpose a large number of calories are prescribed, which are produced from a mixed diet consisting of large quantities of carbohydrates and fats—the albumins in smaller quantities absolutely and relatively.

The other form of lack of internal support is found in neurasthenics and hysterics, more frequently in the former than in the latter. They should be treated generally (see Neurasthenia and Hysteria). Locally it may be necessary to apply the bandage. These patients should be induced to try gymnastics, abdominal massage, and electricity. Postural treatment seems very valuable in this class of cases. They should be taught to lie upon an inclined plane, the head low and the feet high, beginning with slight and ending with great inclination; at first for a short time and then for a longer time.

The results from this form of treatment are excellent, but, do what we may, there remain a certain number of cases in which a surgical operation, nephrectomy or nephrorrhaphy, should be performed. The indications for operation seem to be, at the present time of writing: when the patient's life is endangered, and when the patient is prevented from pursuing his occupation or enjoying his life. The first occurs in the floating kidney, in which there is hydronephrosis; when there is no relief in frequent attacks of Dietl's crises, either nephrectomy or nephrorrhaphy are indicated. Hahn's operation, nephrorrhaphy, should always be preferred on account of its comparative lack of danger. Pedro Chutro thinks that a displaced kidney on the right side usually follows a displaced cecum and that anchoring the cecum to the posterior parietal peritoneum often cures the condition.

ANURESIS

Anuresis is due to failure of the kidneys to secrete urine and must not be confused with retention. It is a symptom not a disease, and may be due to many causes. The treatment depends upon the cause. The chief conditions causing anuresis are: (1) obstruction of the ureter by stone, or more rarely by inspissated pus or blood-clot; (2) acute nephritis, particularly the scarlatinal type; (3) dehydrating infections such as cholera; (4) toxemia, particularly bichlorid of mercury or phosphorus poisoning; (5) chronic interstitial nephritis; (6) marked cardiac decompensation; (7) peripheral irritation, and (8) hysteria. It is to be noted that in the obstructive type both ureters may be simultaneously blocked or, more rarely, stoppage of one ureter may cause reflex anuresis affecting both kidneys.

Treatment.—In the *obstructive type* of anuresis attempts may be made to force on the obstruction by relaxing the patient by means of the hot bath, forcing water internally and applying massage. Such attempts should not be persisted in for more than twenty-four hours and in case of their failure prompt surgical intervention is indicated.

In patients with anuresis due to *nephritis* hot baths, sweats or hot packs should be tried. Fluids should be forced by mouth or should be introduced in the form of the Murphy drip or the subcutaneous or intravenous saline infusion. Heart stimulants may be indicated and diuretics are advised by some authorities. As a final resort Edebohl's operation of stripping the capsule of the kidneys may be tried. It has frequently been successful. In the patients with chronic nephritis venesection followed by saline infusion may be of value.

Where anuresis is due to *infectious processes* like cholera, which act by depressing the circulation and abstracting water, saline infusion and cardiac stimulants (caffein, digitalis) are indicated. The treatment of anuresis due to *toxemia* is considered under the poisons involved.

In anuresis due to *cardiac decompensation* the treatment is directed to the underlying cause. In the *reflex types* the removal of the source of irritation (phimosis, stricture, etc.) is indicated. The treatment of *hysterical anuresis* is very difficult. Decapsulation has been done and even nephrotomy has been tried.

HEMATURIA

The causes of this symptom are treated in connection with the diseases in which it occurs. It will therefore be considered simply as a symptom which should be controlled. The patient should always be put to bed and kept there at least until macroscopic blood has disappeared from the urine. His diet should be unirritating and should contain nothing which may irritate the kidney. When the underlying cause is known, this may not be necessary. At all events, nothing should be given which causes the urine to become irritating. Hemostatics should be administered unless causal treatment can be applied immediately. The remedies which may be employed are (and I give them in the order in which they have been most serviceable to me): adrenalin, calcium lactate, ergot. Pituitrin should be considered because of its excellent tonic effect upon blood-vessels. The other hemostatics should be employed only in milder cases, and in these they have seemed to be useful. I refer especially to tannic and gallic acids and lead acetate.

Nephrotomy should be considered when no cause can be found and the bleeding does not cease. It does not always help matters by checking the hemorrhage; it may, however, be life-saving when the bleeding is checked by it, and, moreover, the cause of the trouble may be found.[2]

HEMOGLOBINURIA

Toxic Hemoglobinuria.—Methemoglobinuria and hematoporphyrinuria are included in our present topic. Hemoglobinuria is found in connection with infectious diseases: syphilis, malaria, scarlatina, typhus, typhoid fever, and yellow fever. It may be the result of purpura or scurvy. It is also caused by a number of intoxications: with acids, such as sulphuric or hydrochloric (chronic intoxication), by the ingestion of solanin (sprouting potatoes), by inhalation of hydrogen arsenic, by the ingestion of uncooked mushrooms, and of soap bark (saponin). In all these conditions there is hemolysis, as there also is in *methemoglobinuria.* This subject has become more and more important as the number of causes has increased, largely on account of the introduction of drugs which produce methemoglobinuria. The clinical picture which existed before the era of new drugs was that of potassium chlorate intoxication, usually fatal but rare. With the introduction of drugs derived from coal-tar, methemoglobinuria has become rather common, and mild cases are recognized, which recover, as a rule. Acetanilid, phenacetin, antipyrin, pyridin, naphthol, pyrogallic acid and carbolic acid and their various combinations are the principal toxic drugs.

---

[2] Hematuria is due in nearly two-thirds of all cases to neoplasm somewhere in the genito-urinary tract. It is very important, therefore, that a thorough urological examination be made promptly in every patient with pronounced hematuria. Symptomatic treatment is only justifiable as a temporary expedient.—Editor.

Hematoporphyrinuria most frequently follows the administration of sulphonal or trional.

It will be seen, therefore, that prophylactic treatment is most important in all these conditions. For hemoglobinuria, everything that tends to reduce the toxins, whether it be antitoxic or antibacillary treatment, should act prophylactically. In other words, specific treatment may prevent this form of blood condition. The trades intoxications can be prevented beyond a doubt; it only wants the will, the way is easily found. Sprouting potatoes should never be eaten.

The prevention of poisoning by drugs which produce methemoglobinuria and hematoporphyrinuria is a serious question, as the intoxication may follow medicinal doses prescribed by the physician. The taking of these drugs habitually and in large doses without the advice of a physician cannot be prevented and does not concern the physician except when he is called to advise treatment. When, however, a physician prescribes a drug and toxic effects follow, although he has done all that should be done, he is blamed for the result. This is perfectly natural, as the layman sees only the direct cause and not the contributing ones which make for the bad result. It is well, then, that the physician exercise great care in prescribing drugs of the class under consideration. Upon the whole it may be said that the antipyrin group gives less trouble than the acetanilid group. The further the derivatives are developed from the mother substance, the milder the effects as a whole. In the present state of drug production it is exceedingly difficult to choose a drug. That one which is recommended by good and, apparently, respectable authority frequently turns out to be very toxic. Physicians not uncommonly feel that new drugs represent advance, at all events that they should prescribe them. The rule should be followed here, as elsewhere, that when a drug fills all the requirements necessary to make it efficacious and safe it should be retained by the practitioner. If he changes to the newer drug he may be changing a bird in the hand for one in the bush, and, what is worse, discarding a tool which fits him perfectly and which he has learned to use as one only can an old tool.

Treatment.—In methemoglobinuria the drug which causes the intoxication should be immediately discontinued. In the acute form of intoxication the treatment can only be symptomatic. Oxygen inhalations are recommended; they may be valuable as not only the hemoglobin but the plasma of the blood as well carries oxygen for its internal respiration. Intravenous injection of normal saline solution should be tried in all the bad cases; among other effects, it dilutes the toxic substances in the blood, producing temporary amelioration which may be sufficient to turn the tide in favor of the patient. Even Sahli's lavage of the blood may be tried. In some instances cardiac remedies are required; caffein seems to be the most frequently used, and should be given hypodermically. It is not rational to believe that when caffein is given with the coal-tar product the cardiac symptoms do not develop. Especially is this true of acetanilid, in which the heart is not so much affected as the respiration. The collapse requires stimulants, camphor, ether hypodermically. The irritation of the kidneys, which comes on early and which may last for some time, should also be treated. In hemoglobinuria due to hemolysis from infections the blood count should be followed daily, and if the process results in severe loss of red corpuscles transfusion should be resorted to.

In hematoporphyrinuria the treatment consists in the removal of the cause, that is, the drug producing it should be removed, and this is sufficient in the majority of cases. But enough cases of poisoning with sulphonal and trional are now on record so that we are better able to meet this emergency than formerly. Sulphonal produces by far the worst forms and the most dangerous; trional poisoning is milder and not so often followed by hematoporphyrinuria. In all these forms of hemoglobinuria there are both acute and chronic forms.

PAROXYSMAL HEMOGLOBINURIA

It is now pretty clearly demonstrated that the two factors of importance in paroxysmal hemoglobinuria are syphilis, especially inadequately treated syphilis, and exposure, either local or general, to low temperature. There are patients in whom anxiety, worry, exertion are followed by an attack. In predisposed women menstruation sometimes causes a paroxysm. Except in the latter case, prevention is simple. Either the patient must be very careful to protect himself against cold, sometimes impossible of accomplishment, or he should move to a warm climate.

Before doing this it is necessary to be absolutely sure that no attacks occur except as the result of cold.

Treatment.—For the treatment the fact must be considered that, in a large proportion of cases, syphilis or a syphilitic history is found, and those who report these cases state that the condition, evidently the cause or a cause, had been removed by antisyphilitic treatment. So much so has this been the case and the therapeutic results so good that the advice is given to put all patients with paroxysmal hemoglobinuria through a course of antisyphilitic treatment. The connection with syphilis can be determined by the existence of a positive Wassermann reaction, so that when this exists active antisyphilitic treatment should be instituted. Even when there is no syphilitic history antisyphilitic treatment has been employed with good results. The employment of the Wassermann test should, therefore, be recommended in all these patients for the purpose of discovering the etiology in the individual case.

There exists an intermittent form of hemoglobinuria which is due to an indirect effect of malaria. The hemoglobinuria which exists in malaria may become intermittent and then resemble the paroxysmal form. In these cases it is well to begin with a course of arsenic, to be followed by quinin. If, for one reason or another, the patient requires prompt relief, quinin must be given as described in the chapter on Malaria. We must never forget that in predisposed people quinin may produce hemoglobinuria, and arsenic seems to prevent it.

During the attack the patient must be kept in bed. The diet should be bland and especially unirritating to the kidneys (see Nephritis). A large quantity of fluid should be taken by the patient so that the hemoglobin may be worked out of the kidneys. This should be done in every case unless there is a possibility of doing harm with it.

Inhalations of nitrite of amyl have been recommended in order to abort these attacks; they may be tried when there is no contra-indication.

The mortality in this condition is very low, although fatal cases are recorded; some patients go about their business unless the urine is very bad and some continue under any condition.

CHAPTER XXIV

NEPHRITIS

JOSEPH L. MILLER

Introduction.—Before undertaking the discussion of the therapeutics of nephritis, it has seemed advisable to discuss in more or less detail the normal and pathological physiology of the kidney. Therapeutics is largely an effort to convert pathological into normal physiology. In the past the pharmacologist has confined his studies largely to the action of drugs on normal animals, and our drug therapeutics is therefore founded largely upon the action of drugs on normal animals. In recent years experimental pathology and experimental therapeutics have attempted, with excellent success, to fill in the gap. Through their work the field of pathological physiology has been greatly extended. They have also furnished us with much-needed information in regard to the action of drugs in pathological conditions—the only state that requires therapeutic measures. Intelligent therapeutics and pathological physiology should go hand in hand. The kidney is an organ which offers special facilities for physiological investigation. It is for this reason that pathological physiology occupies so much space in the discussion of the therapeutics of nephritis.

NORMAL PHYSIOLOGY OF THE KIDNEY

Theories of Renal Secretion.—The physiology of urinary secretion has not been definitely determined. Two theories, each with worthy defenders, must be presented. Ludwig's theory maintains that we have in the secretion of urine a simple process of filtration with the glomerulus acting as the filter eliminating from the blood not merely the water but also the solid constituents of the urine, namely, the inorganic salts and specific elements—urea, etc. To explain the discrepancy between the proportion of the various inorganic salts and especially the urea, in the circulating blood and urine, he assumes that absorption of water and varying amounts of the other constituents takes place in the passage of the glomerular filtrate through the uriniferous tubules. In recent years the adherents of Ludwig's theory have introduced some modifications, claiming now that the absorption taking place in the convoluted tubules is selective in character, so that the theory as now held is not based upon the purely mechanical process of filtration and diffusion. Certain anatomical conditions in the kidney lend themselves to this mechanical theory. The efferent vessel to the glomerules is smaller than the afferent, and for this reason the pressure in the capillary tuft would be higher than in ordinary capillaries. The great length of the urinary tubules might be considered as especially adapted for the reabsorption of the fluid passing through them, but as Stewart states such anatomical evidence is not especially convincing, as the absorption of water is not a function of the seminal tubules, which may be 60 to 70 cm. in length. Furthermore, the physical laws of filtration, diffusion, and absorption, as now understood, cannot account for the process. Serum-albumin and egg-albumin are both indiffusible through animal membranes, yet one passes through the glomerulus without escaping into the urine, and the other is promptly eliminated. Two diffusible substances, as sugar and urea, are present together in the circulating blood. The latter is eliminated as a waste product, the former retained. If, however, the amount of sugar in the blood passes a certain limit, more than 3 parts per 1,000, it is promptly excreted. With a pure filtration theory it is difficult to explain how, in a starving animal, the amount of inorganic substance in the urine may be greatly reduced, while the proportional amount in the blood is only slightly changed. Cushny has recently modified Ludwig's theory and removed some of the objections to it by emphasizing the importance of selective absorption in the tubules, the final composition of the urine being determined by selective absorption. The vital theory of Bowman is based on the selective secretory action of the cells in the tubules, and also a secretory, rather than a filtrative activity of the glomeruli; this theory, however, has relatively few supporters. At present Cushny's explanation is generally accepted.

Secretory nerves for the kidney have not been demonstrated, the secretion being directly dependent upon the rate of blood flow. When the arterial pressure falls below 40 mm. of mercury as following shock, hemorrhage or extreme impairment of the heart muscle, the secretion of urine ceases. Everything which increases the blood flow, as increased blood-pressure or local dilatation of the renal vessels, without reduction in the general blood-pressure, such, for instance, as follows the use of caffein, intensifies the secretion of urine. So far as known, the only way the secretion in the kidney can be affected by the central nervous system is through a local nervous mechanism by means of which the secretory activity is modified by corresponding alterations in the local blood supply. The kidney is a very vascular organ, and it has been estimated that when under the influence of diuretics, within a minute's time an amount of blood equal to its own weight flows through the kidney. This is from four to nineteen times as great as the average supply of other organs in the systemic circulation.

Composition of Urine.—The normal urine in man is yellowish in color, with an average specific gravity of 1.020. The reaction is usually acid, attributed chiefly to the acid sodium phosphates. Folin's investigation suggests, however, that it may be in part due to organic acids. In addition to the water and inorganic salts the most important constituent of the urine is the urea. It is through this channel that almost all of the nitrogen of the body is eliminated, as the amount escaping through the perspiration and feces is comparatively slight. Since nitrogen forms 16 per cent of the protein molecule, the amount of protein broken down in the body can be determined by multiplying the amount of nitrogen eliminated by 6.25. The daily nitrogen elimination varies, but in an average-sized man it would range between 14 and 18 gm., corresponding to 88 and 117 gm. of protein. Of the total nitrogen eliminated, 87.5 per cent, according to Folin, appears in the form of urea. The remaining nitrogen is eliminated in the form of ammonia nitrogen, 4.3 per cent; creatinin nitrogen, 3.6 per cent, and, in the form of purin nitrogen and unknown nitrogen compounds, this latter at times amounts to more than 5 per cent.

Origin and Significance of Urea.—From 30 to 34 gm. of urea are eliminated daily by the normal adult on a mixed diet. As 1 gm. of protein will yield approximately $\frac{1}{3}$ gm. of urea, this would correspond to a protein destruction of 90 to 100 gm. It has been determined that urea is formed chiefly in the liver. Just the exact steps through which the protein molecule is metabolized to produce urea have not been fully determined. It is well known that when ammonia carbonate is added to the blood and perfused through the liver it is converted into urea. After removal of the liver by Eck fistula, the amount of ammonia in the urine is increased; the urea diminished. Urea then arises from conversion of ammonia salts, this process taking place in the liver. As a result of digestion, protein material is broken up into its constituent elements and the nitrogen appears chiefly in these forms: ammonia, monamino acids, and diamino bodies. It has not been definitely determined in what form ammonia appears in the blood, but most likely as a carbonate or carbamate. It is probable that a small amount of ammonia may be converted into urea by the action of other tissues than the liver. Kossel and Dakin have demonstrated the presence of a ferment which is capable of splitting up diamino bodies, one of the end products being urea. The urea arising in this manner is formed largely independent of the liver and probably explains the presence of small amounts of urea in the urine after removal of the liver. Recently Folin and Berglund have expressed the opinion that urea formation is a function of all mammalian tissues and is not predominantly localized in the liver.

The amount of urea nitrogen in the urine varies directly with the amount of protein ingested, while the other nitrogenous constituents, as creatinin, are practically independent of the character of the food, provided it does not contain these bodies when eaten. These nitrogenous compounds are, therefore, formed from the body tissues rather than from the food.

Creatinin next to urea and the ammonia compounds is the most important nitrogenous constituent of the urine. As stated, the amount is independent of the protein ingested, the daily output varying from 1 to 2 gm. In febrile conditions, where we have increased tissue destruction, the creatinin excretion is increased. The purin bodies, uric acid, xanthin, hypoxanthin, adenin, guanin, also arise chiefly from metabolism of body protein, only a small amount arising from purin bodies taken as food. Inasmuch as sulphur is found in most proteins its elimination in the urine may be taken as an index of the amount of protein destruction. Sulphur occurs in three forms, as oxidized inorganic sulphur, conjugate sulphur, and in an unoxidized form as sulphocyanid or ethyl sulphid. Under certain pathological conditions some sulphur may be excreted in an uncombined form.

The chief inorganic salts in the urine are chlorids, sulphates, and phosphates; these arise partly from salts ingested in the food, and partly from the metabolic destruction of body tissues. Of these salts chlorids in the form of sodium chlorid is by far the most abundant, averaging 10 to 15 gm. daily.

Internal Secretion of the Kidneys.—Tigerstedt and Bergman were first to report on the presence of a pressor substance "renin" in kidney extracts. This pressor substance, soluble in normal salt solution, was confined chiefly to the cortical portion of the kidney. It was also soluble in alcohol, non-dialyzable, and destroyed by boiling. Very small amounts, when injected intravenously, caused a rise in the blood-pressure, varying from a few to 25 or 30 mm. The following year Lewandowsky repeated Tigerstedt's experiment, and, although his results were in a manner confirmatory, he, on the whole, was inclined to discredit Tigerstedt's result. Later Livon, Fiori, and Shaw demonstrated a pressor substance in the kidney, while Vincent and Sheers reported lack of uniformity, at times a rise, again a fall, in blood-pressure. Pearce recently repeated these experiments, as he believed the discrepancy in results might be accounted for by kidney extracts of one species being injected into the same or different species. For instance, Tigerstedt used extracts of rabbits' kidney, but at times injected it into a different species. Pearce found that extracts of rabbits' kidney when injected into rabbits, and extracts of dogs' kidney when injected into dogs, failed to show the presence of a pressor substance, although extracts of rabbits' kidney at times gave a slight rise. Extracts of cats' kidney, when injected into the same species, gave quite uniformly a decided rise in pressure, varying from 22 to 34 mm. An extract of rabbits' kidney injected into a dog gave a fall in pressure. An extract of dogs' kidney, when injected into a rabbit, gave a slight rise in pressure. Pearce has also shown that the urine from a normal dog when injected intravenously into a second dog would cause a marked fall in blood-pressure of 40 to 70 mm. Boiling the urine for a few minutes does not destroy this depressor substance, but prolonged heating will cause its disappearance. He was able to precipitate it from the urine by using large amounts of alcohol or ether. Following experimental tubular nephritis, this depressor substance disappears from the urine, but remains in the urine of experimental vascular nephritis.

PATHOLOGICAL PHYSIOLOGY OF THE KIDNEYS

The study of the functional disturbance in the kidney has been facilitated by recent work in experimental nephritis, giving us an opportunity to determine accurately the changes in the character of the urine in the vascular and tubular types of nephritis, and also the action of various drugs upon the secretion of urine in pathological kidneys. It has thrown light on the causes of edema, anuresis, hypertension, heart hypertrophy, and uremia. It has enabled us to study carefully the influence of glomerular changes as contrasted with those of the tubules. At present we have a large number of nephrotoxic substances; the most important are bichlorid of mercury, arsenic, cantharides, chromium, uranium, snake venom, and diphtheria toxins. Some act diffusely, affecting both tubule and glomerulus; others are relatively selective. Uranium nitrate, potassium and ammonium chromate, and bichlorid of mercury affect especially the tubular epithelium, attacking slightly, if at all, the glomeruli. Arsenic, cantharides, and snake venom affect chiefly the glomeruli, with very slight action on the tubular epithelium. Diphtheria toxin has a more diffuse action, injuring both glomeruli and tubules. Recently the strictly selective action of renal toxin for certain structure of the kidney has been questioned; diffuse involvement is the rule. Aschoff reports that Baehr has been able to produce a glomerular nephritis with uranium nitrate. The introduction of any of these substances into a susceptible animal produces a particular type of nephritis with appearance of albumin and casts in the urine.

Extensive granular or fatty degeneration of the tubular epithelium with definite necrosis characterizes the epithelial type. In the glomerular form due to arsenic, apparently, there is a paralysis of the capillaries in the tuft, with little, if any, anatomical changes. However, as shown by physiological methods, serious vascular injury has occurred. Cantharides, on the other hand, produce marked anatomical changes in the glomeruli, desquamation and leukocytic infiltration; and rather marked degenerative changes can also be demonstrated in the convoluted tubules and ascending loop of Henle. Pearce has shown that rattlesnake venom produces still different anatomical changes characterized by extensive exudation and infiltration in both the capsule and glomerular tuft. Tubular involvement is slight or entirely absent.

The study of these various types of nephritis throws some light upon the route through which toxic substances are eliminated from the kidney, as it is probably through the process of elimination that the various degenerative changes are produced. The experimental nephritis described is strictly an acute form. The animal succumbs or complete recovery occurs; whether a real chronic experimental nephritis has ever been produced is questionable. Necrosis of the renal epithelium and some fibrosis were noted by Petroff after prolonged administration of some of the metals. Ophuls later produced a sclerosis of the kidney in dogs and guinea pigs by prolonged feeding of lead carbonate or acetate. He was unable, however, to find at any time albumin or casts in the urine. For this reason it may be questioned whether the condition was actually chronic nephritis. Haven Emerson has produced varying degrees of chronic nephritis by repeated inhalation of alcohol, ether, and chloroform. Uranium nitrate, when given in repeated doses, produces in animals lesions closely resembling the interstitial type of nephritis as seen in man. Even a single acute attack of uranium nephritis may be followed by severe fibrosis, which at times is progressive in character. Squier and Newburgh have recently produced chronic nephritis in rabbits by feeding on a high protein diet. This has actually little clinical significance as the rabbit is a herbivorous animal. Still more recently they have produced in man mild evidence of renal irritation by high protein diet.

As has been stated, experimental nephritis has added not merely to our knowledge of the pathological anatomy of nephritis, but also to the pathological physiology of the kidney as the nature of edema, blood-pressure, cardiac hypertrophy, etc. This phase of the subject will be discussed in considering the nature of the various processes.

Edema.—The edema associated with nephritis is not due to a single causative factor. In the hemorrhagic types the edema may be chiefly or entirely anemic in character. With the chronic interstitial type it is of cardiac origin, and only in some of the acute forms and in chronic parenchymatous nephritis do we have actual renal edema.

The exact cause of renal edema has not been determined. The theories of water, retention, chlorid retention, vascular injury, and changes in the blood, all have their supporters. The only type of experimental nephritis associated with edema is that produced by uranium nitrate, which in the main excites a tubular type of nephritis. Heinek's observation that an animal with chronic nephritis receiving serum from an animal with uranium nephritis develops edema is of extreme interest. Injury to the vessel wall by the serum, in an animal with nephritis, would appear to be the most probable explanation. In fact, recent work leads us to believe that vascular injury is a very important factor. Cohnheim and Lichtheim found that large amounts of normal salt, equal to 92 per cent of the body weight, could be injected into an animal without producing subcutaneous edema, although ascites and dropsy of the various organs readily appeared. When the skin of such an animal was irritated by exposure to the sun's rays or by painting with iodin, typical subcutaneous edema appeared, due, according to Cohnheim, to alteration in the vessel walls by these external agents. Magnus has shown that under the same conditions of transfusion subcutaneous edema appears if the animal has previously received arsenic or a vascular poison, chloral, or chloroform narcosis. It has been shown by Schlayer that in experimental vascular nephritis the renal vessels have lost their power to dilate when excited by certain stimulants, and have diminished permeability. In all forms of experimental edema the introduction of large amounts of fluid is an essential factor. Retention of fluid, due to impaired kidney function, when combined with the increased permeability of the vessel wall, offers a very plausible explanation for renal edema. Pearce, who has studied carefully the question of renal edema, considers three factors are essential for its development—nephritis, vascular injury, and increase in the body fluids. He also determined that the edema is due to disturbed kidney function rather than reduction in the functioning area. The blood-serum of patients with renal edema has a decided lymphagogue action when injected into animals. The flow of lymph, according to Kast, is increased tenfold. These results have been confirmed in a measure by Starling.

The experimental work of Schlayer and Hedinger has thrown considerable light upon one of the factors responsible for edema, for instance, water retention. They tested the various types of experimental nephritis to see if there is any disturbance of the vasomotor control in the kidney, determining the vasoconstrictor power of the renal vessels by electric stimulation and by adrenalin; the vasodilator reaction by the use of diuretics, as sodium chlorid and caffein. Their results indicate that in the vascular type of nephritis (that due to arsenic or cantharides) the vasomotor action of the kidney is disturbed, both constriction and dilatation being impaired; and diuresis fails to appear after the use of sodium chlorid or caffein. With that type of nephritis, where the poison acts especially upon the tubular epithelium, that is, chromium and sublimate, little if any disturbance of the vasomotor control of the kidney has been noted. It is important to remember that in neither of these types of nephritis, when excited by the agents mentioned, is there any edema. Edema only appears after the uranium nephritis, which it is true is of the tubular type. By applying this method of investigation to uranium nephritis, they find that in those cases where edema fails to appear the kidney reacts in the same manner as the normal or as tubular nephritis due to chromium. However, in those animals which develop edema, while the reaction early and late is the same as described, there is a period, usually about thirty-six hours after the injection and corresponding to the time when edema appears, when the kidney vessels react in a peculiar manner. They still constrict and dilate when the proper stimulus is applied. Sodium chlorid and caffein still cause marked dilatation of the renal vessels, but diuresis does not follow. They have endeavored to discover the cause of the impaired permeability. Inasmuch as it does not appear in other forms of tubular nephritis, it can scarcely be due to impairment of the epithelium of the tubules. The disturbance must be, therefore, in the vessels of the glomeruli. We have here apparently an explanation of water retention in nephritis. Edema only appears in the animal experiments when the animal receives a large amount of fluid. When left on a dry diet it fails to develop.

In addition to the lessened permeability of the renal vessels and large intake of water, a third factor is apparently essential. Animals with impermeable kidneys received large quantities of water and salt and still failed to get subcutaneous edema. After waiting four to five days, then repeating the large intake of water and salt, edema developed. Evidently during this interval some change instrumental in the development of edema had taken place, and this Schlayer believes to be increased permeability of the peripheral vessels.

Summary.—Summing up their results, lessened permeability of the renal vessels, increased ingestion of fluids, and increased permeability of the peripheral vessels apparently account for the subcutaneous edema in uranium nephritis.

Pearce, by a somewhat different method, has arrived at the same conclusion. He produced a tubular type of nephritis with chromate, edema, as usual, not developing. He then produced vascular injury with rattlesnake venom, ricin, or arsenic—all well-recognized vascular poisons. To produce a condition of hydremia, the rabbit received through a stomach tube 100 c.c. of water daily. By combining these three factors—renal injury, vascular injury, and increased fluid intake—he was able to cause subcutaneous edema. No one of these alone or any combination of two was sufficient. The nature of this substance, causing injury and increased permeability of the peripheral vessels in nephritis in man, is yet to be determined. In the acute scarlatinal nephritis this increased permeability may be excited either by the retained products of metabolism, the toxins of the invading microörganisms, an internal secretion of the kidney, or in the secretion from some correlated organ. Timofeew has recently explained the vascular injury as due to the products of the destroyed epithelium. This action is not peculiar to the renal epithelium, but may be produced by the dead cells of other parenchymatous organs, and he explains on this basis the interesting edema described by Cohnheim where the clinical picture is that of renal dropsy, but where the urine shows nothing abnormal and autopsy fails to show a nephritis. This peculiar edema has been observed after scarlatina, typhoid, measles, and certain gastro-intestinal disturbances in children. In all of these cases more or less extensive degenerative changes have been detected in the liver or other parenchymatous organs, and he considers the products of this change responsible for the increased permeability and edema.

Widal has laid special stress on the importance of chlorid retention in the production of edema. Clinical experience has shown that restriction of salt lessens the intensity of edema. Experimental work in animals would indicate that chlorid retention does not necessarily accompany edema, and that salt is less essential than water in its production. The cause of the salt retention is still unexplained. It may be due to lessened permeability of the kidney to salt, or to primary retention of chlorid in the tissues. Castaigne's experiments apparently support the latter view. Dogs with experimental nephritis and lessened chlorid elimination were still able to eliminate chlorids in a normal manner provided they were injected directly into the renal artery. He furthermore bled normal and nephritic animals, and then injected an equal amount of normal salt solution. Shortly afterward blood was taken from the renal artery of both animals and its salt content determined. The blood from the renal artery of the nephritic dog contained less chlorid. This would indicate that in nephritis the chlorids are retained by the tissues rather than imperfectly eliminated by the kidney. Martin Fischer's theory of edema has received little support in recent years, either from the physiologist or the clinician.

The truth of Fischer's theory and the value of his therapeutic suggestions are still under discussion, but clinical observation on the whole has failed to support his theory. That a moderate degree of acidosis is present in severe nephritis has been shown by Peabody and others. This is due probably to an inability of the kidney to eliminate the normal amount of acids formed in the body, rather than excessive acid production, although Mosenthal has shown that moderate increased protein catabolism may occur in nephritis and thus possibly account for some slight increase in acidosis.

Increased Blood-pressure.—In both acute and chronic nephritis increased arterial tension is a common occurrence. It is generally conceded that nephritis is one of the definite causes of hypertension, in the parenchymatous and amyloid kidney blood-pressure is not increased, but, in the so-called vascular types, glomerular and interstitial forms, hypertension is the rule. This increased pressure may appear within forty-eight hours after the onset of an acute nephritis.

Hypertension may be present, however, without demonstrable disturbed kidney function. This type is designated as primary or essential hypertension. It is impossible to state, however, that this may not be due to disturbed renal function with failure to eliminate a substance, possibly a product of normal metabolism capable of exciting vascular spasms. The evident relationship between nephritis and hypertension compels us to hesitate to eliminate the kidney as a causal agent in primary hypertension. This type of hypertension is probably due to narrowing of the vessel lumen, due either to vascular spasm or fibrosis, and the renal changes present in late cases are probably secondary to the vascular constriction. The etiology of primary hypertension is still in the theoretical stage. It has been ascribed to excessive protein in the diet, the endocrine glands, focal infections, nerve strain, retention of sodium chlorid and constipation. None of these views has secured any but a limited following on account of lack of facts. Nephritis still remains the sole recognized cause of chronic hypertension.

Experimentally it has been shown that marked reduction of kidney substance by successive operations leads to a permanent condition of high blood-pressure. Whether this is merely the result of reduction of kidney-substance, or from the nephritis excited by the procedure, has not been determined. Uremia is frequently associated with high blood-pressure, and during the acute uremic attack the blood-pressure is often higher than in the free interval. Of extreme interest is the marked variation in pressure in untreated cases of hypertension. Such transitory changes would indicate that a functional disturbance is responsible for the high pressure.

Summary.—Briefly summarizing our present knowledge of hypertension in nephritis: Its exact cause has not been determined. Several theories have been advanced, none of which is free from objection. It is interesting from a therapeutic standpoint that some of these theories assume that hypertension is a compensatory measure; an effort to force more blood through the kidney in order to increase elimination. If this is true, the attempt to reduce blood-pressure may not be good therapeutics. The etiology of primary hypertension has not been determined.

Cardiac Hypertrophy.—Hypertrophy of the heart is common in both the acute and chronic forms of nephritis when associated with hypertension. An increase in blood-pressure of four weeks' duration is sufficient to produce heart hypertrophy. In the majority of cases (82 per cent) Hasenfeld and Hirsch found both sides hypertrophied. Either we are to infer from this that the hypertension prevails in both systemic and pulmonary circulation, or that, as maintained by Pässler, hypertrophy of the right ventricle is due to insufficiency of the left heart with consequent hypertrophy of the right. It is generally conceded that only those cases of nephritis with hypertension lead to heart hypertrophy. It is probable that cardiac hypertrophy, when due to nephritis, is always the result of increased blood pressure. Jores, who has made a study of the frequency of heart hypertrophy in interstitial nephritis, reports that in the primary contracted kidney heart hypertrophy is rarely missed, while in the secondarily contracted kidney it is commonly absent. This is in accord with clinical experience, as, in parenchymatous nephritis, which later passes into a secondarily contracted kidney, the blood-pressure frequently remains normal.

Uremia.—The symptoms of acute uremia can best be accounted for on the basis of an intoxication. The clinical symptoms of nausea, vomiting, diarrhea, delirium, epileptiform convulsions, and coma can scarcely be explained upon any other basis. On account of the wide variability of the clinical picture the question has been raised by Arcoli whether the symptoms can be explained by the action of a constant or single intoxicating agent. The character of this hypothetical poison or poisons is still in doubt. It is generally assumed that the retention in the blood of products normally eliminated by the kidney is responsible for the disturbance. If this were true, we would expect, after removal of the kidneys, a symptom-complex resembling uremia. In animals such removal results fatally within five or six days, yet without the symptoms of uremia. In complete anuresis from obstruction to the ureter, which may persist for ten to twelve days or even longer, before death ensues, uremia is not observed. It might be assumed that the intoxicating agent in uremia is found in such small amount that in obstructive anuresis, or after removal of the kidneys, life is not sufficiently prolonged to permit of the formation of a sufficient amount to produce symptoms. Against this view is the rapid appearance of uremia in certain cases of acute nephritis. Attempts to produce uremia by introduction into the organism of large amounts of urea have been only partially successful. Leiter has produced vomiting, dyspnea and convulsions in dogs by intravenous injections of large amounts of urea (100 to 200 gm.). These experiments are in accord with those of Marshall and Davis. Whether such large amounts of urea are present in uremia in man is questionable. It is reported, however, that the amount present in the tissues of these dogs was not in excess of that found in patients dying from uremia. Hewlett reports malaise and headache after massive doses of urea. It is true that in nephritis the urea is not eliminated in the normal manner; especially in uremia, the increase of urea in the blood over the normal in many but not all cases may be very marked. Frerich, years ago, ascribed uremia, not to urea, but to ammonium carbonate formed from it by the action of a ferment. The absence of increased ammonia in the blood in uremia disposes of this theory. Creatinin and the other nitrogenous products of metabolism have been held responsible, but there is no direct evidence that they play a rôle. Foster and others believe that the various clinical types of uremia may be due to different causes. Where headache and apathy predominate, nitrogen retention is present. Where edema is marked chlorid and water elimination is markedly impaired. In the convulsive type he has isolated a substance from the blood, not urea, with which experimentally he has produced convulsions.

In mild types of nephritis it has been shown that nitrogen elimination is delayed; that is, the interval between ingestion and excretion is considerably longer than in the normal kidney. These mild cases show comparatively little increase in nitrogen in the blood. With severe types of nephritis there is not merely delay, but absolute retention of nitrogen, so that the amount in the circulating blood is greatly increased. The more nitrogen retention, the greater the danger of uremia.

When we study the elimination in nephritis of various other substances normally present we have less evidence that they play a rôle in uremia.

Uric acid and creatin are eliminated in nephritis as readily, as a rule, as the urea. However, the results in this respect are not uniform.

The sulphates and phosphates behave in much the same manner as the nitrogenous products. The chlorids behave differently. In all patients *without edema*, the elimination of chlorids is satisfactory, although there may be marked nitrogen retention and threatening uremia. All nephritics *with edema* show chlorid retention. This is not peculiar, however, to the edema from nephritis, as it occurs in cardiac edema, ascites due to liver cirrhosis, pleural effusions, and in croupous pneumonia.

Retention of fluid in the body is always associated with the retention of chlorids, the water retention being probably secondary. On account of the toxicity of potassium salts, Feltz and Rourke advanced the theory that they were responsible for uremia. They furnished considerable experimental evidence, but later investigators have failed to confirm their results.

Although it has not been proved that imperfect renal elimination is responsible for uremia, the retention theory has the most followers. Ascoli, however, has advanced the theory of nephrolysins. He has shown that an extract of kidney, injected intravenously into an animal, produces albuminuria, and the serum of this animal, injected into a second animal, excites albuminuria. He believes in nephritis the destruction of kidney cells furnishes material for nephrolysins, which are responsible for the uremia. A vicious circle is established by these nephrolysins, leading to further kidney degeneration; so the process becomes chronic. Ascoli and others who have repeated his experiments have never been able to produce a uremia, so the essential part of the theory is still unproved. There is no evidence that the nephrolysins are especially toxic for the nervous system, and, finally, uremia occurs in chronic interstitial nephritis, where little, if any, degeneration of kidney tissue is taking place.

Summary.—Summarizing our present knowledge of uremia, the nature of the toxic agent or agents has not yet been determined. It probably arises from impaired elimination through the kidney, and there is a strong belief, unsupported by positive evidence, that the toxic substance is some product of nitrogenous metabolism.

Value of Functional Kidney Tests.—It has been shown that in nephritis both salts and urea may be eliminated in a normal manner. As a rule, however, nitrogen elimination is delayed and irregular and not dependent closely upon the amount of protein ingested. Urea determination, without a knowledge of nitrogenous intake, is valueless and misleading. Even when the protein intake has been accurately determined the nitrogen output may differ so little from normal that it has little value as a functioning test. Albumin in the urine does not signify nephritis, as it may be present in considerable amount without coexisting anatomical changes in the kidney. Palpation of the kidney usually excites a transitory albuminuria. Small amounts of albumin frequently occur in the urine of apparently healthy individuals.

Spiegler's reagent, which detects albumin in 1:350,000, gives a positive test with practically every urine. With less delicate tests, as acetic acid and potassium ferrocyanid, albumin is detected in only about 4 per cent of healthy individuals. The significance of albumin as an evidence of nephritis depends partly upon the amount present, although as much as 2 per cent has been reported by von Noorden in physiological albuminuria. On the other hand, in chronic interstitial nephritis, albumin may be entirely absent with the ordinary tests, and is never present in large amounts in uncomplicated cases. It is, therefore, impossible, by testing for albumin alone, to determine the presence of organic changes in the kidney.

From the time casts were discovered by Henle in 1841 until the appearance of the centrifuge in 1891, their presence in the urine was considered as positive evidence of nephritis. Since using the centrifuge, it has been determined that casts are commonly present in normal individuals. Gentzen examined a single specimen from 100 apparently healthy individuals, and found granular casts in 36, and hyalin or granular casts in 60. Klieneberger, examining 24-hour collections of urine, found casts in 78.5 per cent. These findings have been verified by many other investigators, so their correctness cannot be questioned.

Exercise increases greatly the frequency with which albumin and casts appear in the urine. McFarland found abundant albumin and casts in six out of seven bicycle riders. After very severe physical labor, as after a boat race, albumin, granular and blood casts are commonly present in the urine. Hence albumin and casts are of much more diagnostic significance when found in urine collected while the patient is at rest. In doubtful cases their presence during a period of complete rest speaks very strongly for nephritis. It is not to be inferred that albumin and casts are of little significance. Especially the presence of granular casts in considerable numbers almost invariably means nephritis.

Rowntree and Geraghty have apparently overcome many of the objections previously made in regard to functional kidney tests by the introduction of phenolsulphonephthalein for this purpose. This method has withstood the test of time better than any previous one and its accuracy has been confirmed since the introduction of the blood urea determinations. The test originated as a result of the researches of Abel and Rowntree upon the action of the phthalein group. A solution is prepared containing 6 mg. of phenolsulphonephthalein to each c.c., and 1 c.c. of this solution is injected into the muscle. For an hour preceding the injection and for two hours following the patient should be induced to drink water freely. At the time of injection the bladder should be emptied, and again at the end of one and two hours. The urine passed at the end of each hour after the injection should be saved separately and labeled. When ready to be examined each specimen is diluted to 1 liter by adding distilled water, and rendered alkaline with sodium hydrate, when it will assume a pinkish tinge. This solution is then compared in a colorimeter with a standard solution. Normally on an average 50 per cent of the phthalein is passed in the first hour, and a total at the end of two hours of 60 to 80 per cent. Lessened and delayed elimination is an indication of impaired renal function. Recently, however, less attention has been paid to delayed secretion, the total amount eliminated in the two or three hours being most important. Simple passive congestion of the kidney may materially interfere with elimination of phthalein. A low phthalein output is extremely suggestive of marked renal impairment. Good elimination, however, does not necessarily mean a normal kidney. Baetjer has shown that, during the stage of acute nephritis, elimination of phthalein may be in excess of the normal, and recently the writer had a patient who immediately after recovery from uremic coma eliminated 50 per cent of phthalein in the first hour. Another patient who eliminated 40 per cent in two hours developed uremia forty-eight hours later, and a patient with 17 per cent in two hours and suggestive uremic symptoms cleared up. Mayo reports 2 cases with an output of 70 per cent where uremia developed after an operation. This, as other functional tests, often shows very little variation from the normal in chronic interstitial nephritis. The simplicity and accuracy of this test make it, however, the most satisfactory functional test developed up to the present.

Recently the determination of the blood urea has assumed great importance. This procedure is only available for those having access to a properly trained laboratory worker. Inasmuch as the results obtained coincide quite closely with those obtained by the very simple phthalein test, the latter still retains its value. The normal amount of urea nitrogen in the blood varies considerably, according to Folin from 8 to 15 mg. per 100 c.c. of whole blood. Some have placed the upper limit of normal as 20 mg. In nephritis this may be greatly increased. The presence of

100 mg. is suggestive of impending uremia. As before stated, there is not a marked increase in some cases of uremia. Foster reports one patient with 39 mg., and we have seen typical uremia with less than 40 mg.

The determination of blood uric acid and creatinin in nephritis is of much less importance. Creatinin determination is really only indicated when the urea nitrogen is found to be very high. The presence of 5 mg., or more of creatinin per 100 c.c. of blood is an unfavorable prognostic sign, and in the majority of cases points to an early fatal termination. It is interesting to note, however, that fluctuations in blood nitrogen have little, if any, effect on blood-pressure, and evidently the hypertension is not due to retained urea. The determination of the non-protein nitrogen in the blood has given us an accurate index of the ability of the kidney to eliminate nitrogen and the degree of accumulation in the blood that has occurred. The phthalein test shows the functioning capacity of the kidney only during the two or three hours of the test, the non-protein nitrogen determination the accumulated incapacity of the kidney.

The renal test diet is of considerable value in the study of renal function, as it is intended to show how the kidney will behave under slight strain. The test is for water, nitrogen and chlorid elimination, and the test as shown by Mosenthal may be greatly simplified and still retain much of its value (for a full description of the method see *Archives of Internal Medicine,* xvi, 733, 1915).

PRINCIPLES OF TREATMENT OF NEPHRITIS

The treatment of nephritis may be considered under the head of prophylaxis, general principles, symptoms, and complications. The general principles include, first, to avoid as far as possible everything that might tend to further injure the kidney; second, as the kidney function is impaired, to relieve it of excessive work. This is to be accomplished by proper diet and by calling upon the skin and bowel to assist in the elimination of water and solids; third, the use of such measures as would tend to restore the kidney to its normal physiological function.

The treatment of nephritis is chiefly symptomatic. There are no specific remedies capable of modifying the degenerative processes in the kidney. Our efforts in the acute forms must be devoted toward relieving the kidney of its work and removing, if possible, the cause of the trouble, thus tiding matters over until the disease subsides and the physiological function of the kidney is wholly or partially restored. This restoration of function may be brought about in several ways. The acute process may subside and regeneration of destroyed tissues takes place, or the normal kidney tissue undergoes hyperplasia, or the parenchymatous nephritis passes into a secondary contracted kidney, with increased urinary output of both water and solids—a restoration in part of the disturbed function. The hypertension and cardiac hypertrophy developing in interstitial nephritis lead to increased elimination through the kidney, and may be considered a compensatory process. Such a patient may live for many years without any evidence of acute renal intoxication, the symptoms complained of being entirely the result of the hypertension. So complete is the restoration of function in interstitial nephritis that death is rather infrequently due to uremia, but to ruptured arteries or incompetent heart, the result of prolonged high blood-pressure. In these cases treatment must be directed toward so regulating the manner of living as to avoid overtaxing an already overworked heart.

The treatment of a chronic interstitial nephritis is chiefly directed toward the heart. In chronic parenchymatous nephritis the heart is not usually involved and the treatment is confined to the kidney. An important part of the treatment is to avoid all those things, either in the form of food, chemicals, or habits of the patient, that might irritate the kidney; to arrange a diet with the minimum amount of protein consistent with the demands of the body, and to supply carbohydrates and fats in sufficient quantity to maintain the proper nourishment of the individual, a point quite as essential as the restriction of proteins. To facilitate elimination through the skin a warm climate and suitable clothing are important. The bowels should be kept free to assist in the elimination of water, salts, and nitrogenous waste. As the individuals are usually anemic and undernourished, too free catharsis must be avoided. For the anemia induced by the continued loss of albumin, iron or foods containing iron should be given. Suitable foods, however, are of much more value than preparations of iron. To lessen the edema restriction of fluids and salt is important. All of these measures are also indicated in the treatment of acute nephritis. With the kidney acutely involved, extreme precautions must be taken to prevent further injury.

Prophylaxis.—The chief etiological factors in nephritis are the acute and chronic infections, chemical poisons, either exogenous or endogenous, cold, and pregnancy. Certain of the acute infections are specially liable to induce nephritis, as scarlatina, and the utmost care should be exercised by the parents to protect their children from contracting it. The importance of exposure to cold, in determining the development of scarlatinal nephritis, has been probably overestimated. The bacterial toxins are, no doubt, the exciting cause, although cold may act as an important contributing factor. Tonsillar infections are another important source of acute nephritis, and the child subjected to repeated attacks of tonsillitis should have the tonsils removed. It is unwise, as a rule, to remove infected tonsils during the very acute stages of nephritis. Removal of the tonsils may lessen the danger of contracting diphtheria, which next to scarlatina is the acute infection most liable to be complicated by nephritis. It is questionable whether the removal of teeth and tonsils is of any special value in the treatment of chronic nephritis. Certainly the indiscriminate removal, as now practiced, merits condemnation. If a nephritic has diseased tonsils they should be removed. There is no reason, however, to interfere where there is no past history of tonsillitis, unless it can be shown the tonsils are serving as a nidus for the growth of bacteria. As cultures of hemolytic streptococci can be obtained from all normal tonsils, the securing of positive cultures cannot be advanced as an indication for removal. Even more reprehensible is the indiscriminate removal of teeth, because it cannot be said that they are functionless.

There has been considerable difference of opinion in regard to the rôle of syphilis in acute nephritis. It may be extremely difficult to determine in a given instance whether mercury or the syphilis is responsible for the trouble. However, we have authentic cases where syphilis was the etiological factor. At least, the prompt disappearance under treatment would lead to that inference.

There is a long list of chemical agents, many of which are frequently prescribed, which are capable of irritating the kidney and setting up an acute nephritis. It is important in the treatment of nephritis that drugs of this sort be avoided. It is also advisable in those acute infections liable to kidney complication that these agents be used with care.

Attention has already been called to those substances used to excite acute nephritis in animals—cantharidin, chromium, bichlorid of mercury, and arsenic. In addition a number of drugs are extremely irritating to the kidney and capable of exciting acute degenerative changes in the kidney in man. The more important are potassium chlorate, salol, carbolic acid, turpentine, salicylates, lead, phosphorus, alcohol, chloroform, and ether. Even sodium chlorid and theocin in large amount may excite nephritis in some of the lower animals. Especial importance is attached to the rôle played by mercury in nephritis. The readiness with which grave kidney changes may be produced in the lower animals by the use of bichlorid impresses us with the importance of carefully watching the urine when mercury is used and the danger attending its use in patients with nephritis. Klieneberger has made a careful study of the urine in both syphilitics and non-syphilitics during inunction cures, and calls attention to the frequent appearance within four to six days of numerous hyalin casts and their persistence for several weeks after the treatment is discontinued. Hyalin casts are not usually considered of great significance, but their frequent appearance and occasional association with red blood-corpuscles and very rarely with albumin, in Klieneberger's cases, are certainly highly suggestive.

Although arsenic will produce a nephritis in animals, man tolerates it very well. Arsphenamin from present reports, is apparently only mildly irritating to the kidney, as only occasionally have albumin or casts been reported after its use.

Both experimentally and chemically the salicyl compounds have been shown to irritate the kidney with appearance of albumin and casts (Lüthje, Klieneberger, and Oxenius). Klieneberger reports that the administration of alkalis will prevent the kidney irritation of the salicyl compounds. Salol is still more irritating and numerous cases of nephritis were observed as a result of the Woodbridge treatment of typhoid, in which salol played an important rôle. Where the surface of the skin is broken, the local application of remedies as iodoform, mercury, turpentine, pyrogallic acid, balsam of Peru, and tar preparations should be undertaken with care on account of the readiness with which they may set up an acute nephritis. The application to large surfaces of the body of turpentine, kerosene, or pyrogallic acid may cause an acute nephritis. Ether and chloroform narcosis may injure the kidney. The danger can be largely eliminated by skill in administering the anesthetic. The danger of nephritis from anesthesia is always greater when the kidney is irritated by some existing acute infection, as, for instance, operations for some of the complications of scarlatina. Here the anesthesia should be as brief as possible and the minimum amount of anesthetic used. There is no reliable evidence that large meat eaters are especially prone to contract nephritis. The same is true of those eating freely of irritating or highly seasoned foods.

Endogenous toxins arising in the intestinal canal, the result of food decomposition or bacterial products, have been considered by some as important factors. Here again, however, we lack definite evidence.

Prolonged exposure to cold and, especially, wet cold, should be avoided. Siegel has shown that dogs allowed to stand for ten minutes in ice water and then placed in a cage without drying developed typical acute parenchymatous nephritis. The urine contained albumin, casts, blood and renal epithelium, and anatomically marked degenerative changes in the kidney could be demonstrated. When the dog was placed in the cold water and then thoroughly dried before placing in the cage nephritis did not develop. The manner of action is not clear. It has been assumed by Wertheimer that the external cold constricted the renal vessels, this ischemia leading to degenerative changes.

We have too little knowledge of the agencies responsible for the kidney of pregnancy to discuss intelligently prophylactic measures. To refrain from using drugs that may act as a renal irritant and avoid exposure to wet cold is advisable. Although comparatively little is known of the importance of diet in the toxemia of pregnancy, it is advisable in threatened cases if the blood urea is high, which is rarely the case, to reduce the proteins to the minimum consistent with maintaining the proper nutrition.

The appearance of albumin and casts after severe athletic exercise, as racing, raises the question whether severe exertion might set up a nephritis. It is doubtful whether such exercise, even when frequently repeated, can cause nephritis. However, in those people with hereditary tendencies such severe exertions should be avoided, as should also occupation where there is exposure to lead, mercury, and turpentine, or to the extreme changes in temperature, as, for instance, in cooling rooms.

Climate.—The patient suffering from acute nephritis is better off at home, provided he can have proper attention. The danger of overexertion or exposure to cold renders traveling undesirable. Change of climate is usually only to be considered in the subacute or chronic forms. The chief object of such a change is to place the patient where a warm and dry climate dilates the peripheral vessels and increases the elimination through the skin. In such a climate the patient is enabled to be out of doors, in the sunshine and fresh air, factors which perhaps have only a psychological effect, yet nevertheless are of considerable importance. The patient with parenchymatous nephritis is often free from uremic symptoms in temperate climates during the summer months. With the approach of cold weather he often becomes distinctly more uncomfortable. The effect of cold is still more marked in patients with interstitial nephritis and high blood-pressure. During the cold days of late autumn they notice increased poststernal distress on walking, or this may be increased to the point of actual pain in the form of angina pectoris. They are more liable to attacks of nocturnal pulmonary edema, especially if they dress or undress in a cold room. The poststernal distress is probably due to the increase in blood-pressure on a heart already overtaxed, by the constriction of the peripheral blood-vessels from the cold.

The chief benefit of change of climate for the patient with parenchymatous nephritis is to increase the elimination through the skin. The daily loss of water through this channel may be much greater in a dry warm climate than we are able to induce by artificial sweats. Loew has shown that the daily loss of water through the skin and lungs of a resting patient, in a dry climate like southern California, may reach 2 to 2½ liters (2 to 2½ quarts). It is true that the amount of solids in the perspiration is not very great: about 1 gm. (15 gr.) of urea and about the same amount of sodium chlorid per liter. However, it is quite certain that under these conditions the chronic uremic symptoms become less marked. When possible it is advisable to select a climate where the nights are not too cool. This is, however, difficult, and may be easily overcome by suitable clothing and having the patient remain indoors after sunset. Altitudes above 3,000 feet should be avoided, especially by those patients with weak hearts or high blood-pressure. Patients with hypertension, who are able to walk about, should avoid hilly regions, as this may materially interfere with their out-of-door exercise. If the place selected is on the seashore, sea bathing should be avoided, as the cold and exertion are both undesirable. In this country a suitable winter climate may be found in southern Texas, southern California, the low altitudes in Arizona, New Mexico, and Mexico. The climate of the Gulf States is less desirable on account of the greater amount of moisture. Abroad, Egypt, Sicily, southern Italy, as Capri, Sorrento, Cannes, Mentone, and San Remo. Abroad, a suitable summer climate at moderate altitudes can be found in the Black Forest. In this country during the summer, the patient does best in a climate where the nights are comfortably cool as in northern Michigan or Wisconsin, and along the northern seashore. It has usually been considered that high altitudes are dangerous to patients with high blood-pressure. Recently Staubli has made some observations on the effect on hypertension in patients at St. Moritz, Switzerland. He reports that continued residence at such an altitude is followed by a rather decided fall in blood-pressure with gradual return to the previous level after the patient goes back to a low altitude. The patient during his residence in the high altitudes feels better, getting relief from certain cardiac disturbances, as angina pectoris, poststernal distress, and palpitation. The question arises how much of the improvement is due to high altitude per se, and how much to mental relaxation and psychical effect of a visit to a place like St. Moritz. Staubli's observation shows that the dangers of high altitude for this class of patients may have been overestimated. Schneider, in a recent investigation, reports that when at rest, in high altitudes, blood-pressure is lower than at the sea level, but on exertion the quickening of the pulse and increase in blood-pressure are more marked and prolonged than at lower levels.

Climate does not cure the chronic forms of nephritis, but it makes the patient more comfortable and not infrequently prolongs his life by preventing acute exacerbations and lessening the tendency to uremia.

Rest.—The patient with acute nephritis should have absolute rest in bed. This insures uniform warmth to the surface of the body, limits body nitrogenous waste to the minimum and reduces the work of the heart. Standing or exercise increases the amount of albumin and number of casts. The increased nitrogenous waste with moderate exercise is almost imperceptible, and is of little importance. The rather decided rise in blood-pressure frequently observed very early in the glomerular type of the acute nephritis increases very materially the work of the heart. Rest until the necessary hypertrophy has taken place is important. Mental strain raises blood-pressure and should be avoided.

The patient with chronic parenchymatous nephritis should observe quite closely the same precautions in regard to rest as the individual with the acute form. When edema is present little exertion should be indulged in. To obtain fresh air, sitting out-of-doors or riding in suitable weather may be permitted. At all times protection from the cold must be observed. To carry out such precautions for months or even years is often difficult, but it is important if we wish to avoid the acute exacerbations so commonly seen in this type of nephritis. The benefit derived from prolonged rest in bed is frequently observed in patients in a free public hospital, where they may remain in bed or at least indoors and quiet for months or even a year or more, with gradual improvement. As a rule, when such a patient leaves the hospital he will return within a few weeks with all the old symptoms and another prolonged stay in the hospital is necessary. Rarely does a patient kept quiet in this manner suffer from the acute exacerbations so commonly observed in the parenchymatous type of nephritis. In the chronic interstitial nephritis the same rules as regards rest should be observed as are commonly employed in weak hearts. On account of the increased work thrown upon the heart by the high blood-pressure, overtaxation should be carefully avoided. Just the degree of rest is dependent upon the working power of the heart. Where decompensation is marked, absolute rest in bed may be necessary. In other cases it is only advisable to avoid hill or stair climbing, or heavy lifting. A safe guide for the patient is the appearance of dyspnea, and he should be taught to avoid all those things which cause shortness of breath. It is much easier to prevent overtaxation and dilatation of the heart than it is to restore it again to its previous condition. When cardiac insufficiency develops, the result of hypertension, the probability of lasting improvement is not very great. In many of these cases the high blood-pressure can be very slightly or not at all modified, and it is very difficult to restore a heart which is constantly compelled to do increased work. Massage may be used advantageously in the chronic form of nephritis. Where the heart is weak it should be given lightly, and when it excites fatigue or cardiac disturbance should be discontinued or given less vigorously. The use of fats, as cocoa butter, commonly employed, should be avoided, as they interfere with proper elimination through the skin.

Bathing and Care of the Skin.—Proper functioning of the skin is very important in all forms of nephritis. To maintain this, frequent bathing is essential. In nephritis prolonged cold baths are contra-indicated, as cold temporarily constricts the peripheral vessels, causing hyperemia of the splanchnic circulation, including the kidney, and also interferes with the secretion from the skin. With interstitial nephritis the cold contracts the skin vessels and raises blood-pressure. When given, except for the purpose of sweating, baths should be as near the body temperature as possible. There are no special advantages to be derived from medicated baths.

Elimination through the Bowel.—Good elimination through the bowel is essential in all cases of nephritis. With impending uremia or edema very free catharsis is important. By free catharsis the amount of nitrogen eliminated through the bowel may be greatly increased.

While normally 10 per cent or less of the nitrogen ingested leaves the body in the stool, in uremic patients with diarrhea this may reach 30 per cent (von Noorden, Ritter). This vicarious action of the bowel becomes, therefore, of considerable importance. The normal daily feces contain less than 1 gm. of sodium chlorid. By means of free catharsis this may be increased to 4 or 5 gm. daily, a matter of considerable importance in those cases where the kidney is impermeable to salt. The chief value of free catharsis, however, is to get rid of fluid, and it is especially indicated where edema is present. Judgment must be exercised when employing this means to dispel dropsy. The patient may be quickly freed of his dropsy by excessive catharsis, but left in such an exhausted state that his condition is more serious than before. Especially prolonged catharsis may do harm by impairing nutrition. With proper restriction of fluids and salt, and with a low-nitrogenous diet, extreme catharsis is rarely required. The cathartic selected should produce a watery stool, and at the same time not irritate the kidney. On account of their irritating action, mercury preparations should be avoided. Calomel, however, on account of its diuretic action, is often employed. A saline cathartic is usually quite as efficacious as calomel, and safer. In selecting a saline cathartic palatability should be considered. Sodium phosphate in doses of 4 to 20 gm. (1 to 5 ʒ) dissolved in hot water is reasonably palatable. Calcined magnesia either in the powder form in 1 to 4 gm. (15 to 60 gr.) or in the form of milk of magnesia, 15 to 60 c.c. (½ to 2 ℥), is usually well borne. The liquor magnesia citrate (U. S. P.), while reasonably palatable, is somewhat objectionable on account of fluid contained in dose, 360 c.c. (12 ℥). The sodium and magnesium sulphates, while efficient, are extremely distasteful to most patients. Some patients prefer some of the bottled saline waters, as Apenta or Hunyadi. Saline waters containing large amounts of sodium chlorid should be avoided where there is evidence of salt retention. The effervescing salines, as effervescing phosphates, or Seidlitz powders, while palatable, are objectionable on account of the amount of gastric distention. This distention is especially dangerous in dropsical patients with ascites, where the stomach is compressed below, and the enlargement forces the diaphragm upward. The salines are best taken in the early morning on an empty stomach, and preferably in half a glass of hot water. The milk of magnesia, on account of its palatability, may be taken at frequent intervals during the day.

Of the vegetable cathartics, jalap and elaterium are most frequently recommended on account of producing a watery stool. Jalap is usually given in the form of compound jalap powder, pulvis jalapæ compositus (U. S. P.), in dose of 1 to 4 gm. (15 to 60 gr.). Elaterium may be given in pill form in dose of 10 mg. (⅙ gr.), or as elaterin, 2 to 4 mg. (1/30 to 1/15 gr.). Both jalap and elaterin are more apt than the salines to cause griping. Any of the other vegetable cathartics, as cascara sagrada, rhubarb, aloes, or senna, may be used, but are less desirable, as they do not produce a watery stool.

Restriction of Fluids in Nephritis.—Until comparatively recently it has been the custom to give patients with nephritis large amounts of fluid. In the acute and chronic parenchymatous types it was thought by this means to flush out the kidney and free the secreting tubules of casts, epithelial cells, and other débris. Toxins were by this means eliminated in a more dilute form and rendered less harmful to the kidney. It has been shown, chiefly through the work of von Noorden and his associates, that in certain forms of nephritis, water drinking may be harmful rather than beneficial. With the dissemination of von Noorden's views, many have concluded that restriction of fluids is indicated in every case of nephritis. It is necessary, however, to discriminate, not merely in regard to the class of cases where such restriction is indicated, but also the amount of fluids allowable.

In every case of nephritis where the ability of the kidney to eliminate water is seriously impaired, as shown by the presence of renal edema, restriction of fluids is indicated. The ingestion of large amounts of fluids under these circumstances only increases the edema. In some patients, where edema is absent or at most very slight, the ability of the kidney to eliminate water is limited. Such a patient, if given a large amount of fluid, will develop an edema. It is important, therefore, to determine the relation between fluid intake and output. The most satisfactory method of determining this is by changes in the patient's weight. Simply measuring the fluid intake and comparing this with the fluid output, as determined by the amount of urine plus the stool in case this is watery, is far from accurate. The actual fluids can easily be measured, but to estimate that taken in the solid foods is difficult. In addition, water is formed in the oxidation of foods; 100 gm. of fat in the splitting-up process gives rise to 41 c.c. of water; the same amount of protein and carbohydrates gives 118 and 35 c.c. respectively. Roughly the water formed in this manner plus that in solid foods approximates 700 c.c. A considerable amount of water is eliminated through the skin and in the expired air. Each of these show wide variation, but approximately a total of 800 c.c. is eliminated daily through these channels.

The daily excretion of urine in chronic interstitial nephritis is in excess of the normal, and when edema appears, it is usually of cardiac origin. In this type of nephritis the kidney has lost its ability to excrete a concentrated urine. Its permeability to water, however, is normal. It is important in these cases to give a sufficient amount of fluid to carry off the requisite urinary solids. It is desirable to determine approximately the amount of fluids required, rather than attempt to flush out the kidney by large amounts, and thus still further tax an overworked heart. Mohr has worked out this phase of the subject, and his results furnish us with a safe guide. He determined that in any form of nephritis practically the maximum amount of solids was eliminated if the patient passed from 1,250 to 1,500 c.c. of urine. Von Noorden recommends in chronic interstitial nephritis, when restricted to this amount of fluid, to give once a week a flushing out day, to carry off any retained waste. On this day the patient may take 3,000 c.c. of fluids. Patients with severe renal edema may require much less fluid than the above-mentioned amount. The requisite quantity should be determined by studying each case in order to determine the permeability of the kidney to water. If by administering water there is not a corresponding increase in the urine we are simply increasing the edema. Patients with uremia and marked edema may be given advantageously only a pint of fluid in the form of milk or cream daily, until the urinary flow is increased. Then gradually increase the fluid intake, but not exceeding 1,500 c.c. daily. Recently Williams and the writer studied the effect of taking large amounts of fluid in 3 cases of nephritis with nitrogen retention. By means of the Rehfuss tube we were able to administer 8 to 10 liters of water daily. This was continued for a week, but without reduction of the blood urea. The effect on blood-pressure was also observed. When 2½ liters were given in a period of fifteen minutes the systolic blood-pressure rose from 15 to 40 points. It is very doubtful, however, whether free drinking of water will have any effect on blood-pressure, and from this standpoint restriction of fluid is not important.

There are no advantages in special waters. Pure water has all the advantages of the more expensive medicated forms. The popular belief that patients with nephritis may be benefited by going to certain springs and drinking freely of the waters is fallacious.

Summary.—The maximum amount of waste can be eliminated through the kidney, if the urinary output approximates 1,500 c.c. daily. Patients with marked renal edema should take, as a rule, not more than 1,000 c.c. of fluid daily. If the urine is very scant, and edema marked, half of this amount may be given. The presence of renal edema is evidence of inability of the kidney to eliminate water. To administer large amounts of fluids simply increases the edema.

Restriction of Sodium Chlorid.—A few years ago Widal and Javal called attention to the inability of many nephritics to eliminate sodium chlorid in a normal manner. The salt retained in the tissues requires a certain amount of water to maintain it in the proper molecular concentration, thus leading to edema. Widal considered that chlorid retention was responsible for the dropsy in nephritis. In some cases of nephritis the kidney was permeable to urea, phosphates, and sulphates, but not to chlorids. For this reason he considered the chlorid elimination a better index of the functioning power of the kidney than the excretion of urea. Although Widal probably overestimated the importance of chlorids in edema, his teachings have been very valuable, as there is no question that in many cases edema may be reduced by the restriction of sodium chlorid. It is only necessary to weigh a patient with renal edema, who has been partaking freely of salt, then place him on a salt-free diet and notice the rapid loss in weight. Not every patient with parenchymatous nephritis shows chlorid retention, and many normal individuals have very marked inability to excrete chlorid, and after the ingestion of quantities of salt show a marked gain in weight. This retention may be quite equal to that observed in mild nephritis. Chlorid retention is only marked in severe acute or chronic parenchymatous nephritis. In the milder forms and in the interstitial type chlorid elimination approaches the normal. O'Hare has shown that as a rule salt and water elimination is disturbed earlier than nitrogen excretion.

Widal also considered that the chlorids had considerable influence on the degree of albuminuria and the tendency to uremia. The observations of Blookers and others have failed to confirm this. Blookers' patients did not show an increase in the albumin when sodium chlorid was given freely. The amount of chlorid consumed by the average individual is far in excess of the amount required by the body. Two, or at most 3 gm. is all that can be utilized, and any in excess of this amount is promptly eliminated. The amount taken daily on the average is 6 to 10 gm. A safe rule to follow in nephritis is to give the patient only the amount of salt demanded by the tissues, for instance, 2 to 3 gm., simply avoiding the excess. It is rarely, if ever, advantageous to reduce the chlorids below this point, and certainly never for long periods of time. This can be accomplished by avoiding meat soups which may contain 6 to 8 gm. of sodium chlorid per liter. Their high chlorid content, the presence of extractives, large amount of water and small amount of nourishment render them especially undesirable. Salt should not be added to the food, except to butter and the small amount present in the bread. If the patient does not object to unsalted butter, this may be used. If meats are permitted, they should be fresh, not salted. If the patient's appetite is poor, to render food unpalatable by withholding salt may do more harm than good. There is, on the other hand, little danger of the patient receiving less than the physiological requirement, as 1 liter of milk contains almost 2 gm. of salt, which gives the patient all he needs. The tables on page 533 show the salt content of some of the commoner articles of food. These refer to the amount of salt naturally present. The figures are taken from Strauss' book on dietetics.

Summary.—In all forms of nephritis associated with renal edema restriction of sodium chlorid to 2 or 3 gm. daily is desirable. This amount gives the patient his full physiological requirement. A salt-free diet is only necessary where edema is marked, and should not be main-

Sodium Chlorid Content of Various Foods

Food	*Percentage of NaCl*	*Food*	*Percentage of NaCl*
Milk	0.16	Blueberries	0.02
Butter, salted	1.00	Apples	0.06
Butter, unsalted	0.02	Oranges	0.04
Cheddar cheese	1.01	Oatmeal	0.16
Edam cheese	2.57	Rice	0.10
Cream cheese	2.00	Buckwheat	0.06
Holland cheese	2.60	Nestlé's food	0.29
Eggs	0.14	Barley	0.40
Caviar, Russian	6.70–7.2	Radishes	0.02
Lean beef	0.10	Celery	0.31
Oysters	0.59	Cucumbers	0.07
Fresh fish	0.10	Spinach	0.21
Ham, raw	4.10–5.8	Potatoes	0.06
Ham, cooked	1.80–5.3	Mushrooms	0.06
Corned beef	2.04	Cabbage	0.08
Mackerel, salted	11.42	Lettuce	0.13
Liebig's extract	3.49	Asparagus	0.83
Peas	0.06	Artichokes	1.27
Beans	0.05	Tomatoes	0.14
Cherries	0.01	String beans	0.83
Grapes	0.01	Plums	0.003
Gooseberries	0.05	Apricots	0.004

Amount of Salt Contained in the Average Portion of Food for One Person As Usually Prepared

Food	*Grams*	*Food*	*Grams*
Filet of beef	3.10	Potato soup	1.38
Roast beef	2.80	Spinach	2.90
Liver	4.10	Cabbage	0.50
Roast pork	2.30	Mushrooms	0.50
Cooked fish	4.00	Lettuce salad	0.78
Bouillon	2.60	White bread, 100 gm.	0.40–0.7
Cream soup	0.70	Rye bread, 100 gm.	0.75
Cereal soup	1.85	Zwieback, 100 gm.	0.38

tained for a prolonged period. Chlorid restriction is not necessary in pure chronic interstitial nephritis, as renal edema is rarely present. Sodium chlorid does not increase the albuminuria.

Sweating.—There can be no question of the value of sweats in certain forms of nephritis. It is probably true that the benefit derived is not entirely dependent upon the amount of water and solid material eliminated. Many patients with cardiac dyspnea, due to high blood-pressure arising from interstitial nephritis, are greatly relieved by profuse sweats. There may be no evidence in such a patient of renal edema, and the elimination through the kidney may be normal. Here the relief obtained is due to dilatation of the peripheral vessels thus relieving the heart.

This lowers blood-pressure, thus relieving the left heart of its excessive work, and the dyspnea disappears. For the cardiac dyspnea often appearing in the evening or during the night, due to hypertension, there is no other measure that so frequently gives such striking relief as sweating. In these cases the sweat should be given a short time before the usual hour at which the dyspnea appears. Daily sweats often favorably affect the high blood-pressure. After the sweat, the patient should not be exposed to the cold. In the case of a patient of mine in a hospital, the attendant in the hydrotherapy room, who had been accustomed to treat chiefly nervous patients, turned on the cold spray, causing the patient to faint. The day following, the same procedure was gone through with. The patient's systolic blood-pressure, previous to the sweat, was 220; after the sweat it was 180. When the cold spray was applied it promptly rose to 260.

The amount of water lost in a profuse sweat may be as much as 1 liter (Kovesi, Strauss). Kovesi has shown that there is included 8 to 9 gm. of solid matter; 2.86 gm. of which was sodium chlorid and 2.08 gm. nitrogen. Strauss studied the perspiration in 63 cases, chiefly nephritis, and found the amount of sodium chlorid varied from 0.3 to 0.7 per cent. The amount of water varied from 200 to 800 c.c. With the latter amount, if it contained 0.7 per cent sodium chlorid, the patient would eliminate in the sweat 5.6 gm. This amount is, however, far above the average, which is not more than 2 gm. Strauss found the amount of nitrogen in the sweat varied from 1 to 2 gm. per liter, about one-third of which was urea nitrogen.

Leube and others have expressed a fear that in withdrawing such a large amount of fluid from the body, the increased concentration of those substances exciting uremia might precipitate an attack. This fear is not supported by clinical experience. Strauss, who has had very extensive experience, has never seen evidence of increased intoxication after a sweat. Inasmuch as the percentage of urea in the perspiration is considerably higher than in the blood (Plaggemeyer and Marshall) there can be little danger of exciting acute uremia by sweating.

Regarding the danger of shock from the marked dilatation of the peripheral vessels, there is little to be feared, especially if the patient is lying down when taking his treatment. It is advisable when the first sweat is given to have the physician present. After this, any intelligent person can safely supervise it. If the pulse becomes poor, or the patient complains of faintness, the heat should be discontinued and the surface of the body bathed with alcohol. The degree of cold produced is sufficient to constrict the skin vessels and in so doing restore balance to the circulation.

Various methods may be employed for inducing the sweat. The chief consideration is to use a means which will produce the most profuse perspiration with the least inconvenience to the patient. In hospitals or institutions with hydrotherapeutic facilities an excellent method is to place the patient in one of the large ovens used for the purpose. A cold towel should be applied to the head and the sweat continued for twenty minutes. The patient may then be removed from the oven, wrapped in blankets and allowed to cool gradually, or what is more comfortable is to rub the patient off with alcohol, avoiding chilling of the body and then cover him up warmly with blankets, these to be gradually removed.

In the home, either the hot pack or hot-air bath may be used. The former does not require any special apparatus and the results are satisfactory. A dry sheet and blanket are placed over the patient, hot-water bags or bottles or hot bricks placed along the sides of the trunk and extremities and then several blankets carefully tucked in and spread over all. A cold cloth should be applied to the head. It may be necessary to renew the hot-water bottles, but, as a rule, this is unnecessary. The average duration of the sweat is twenty to thirty minutes. Care must be taken to protect the patient from burns. The hot-air bath may be either dry air or steam. The patient in bed is covered over with blankets, which must be supported on a frame so as to allow free space for the hot air to circulate. If a steam bath is to be given, a teakettle, with an alcohol lamp, gas, or electric heater, is placed on a chair at the foot of the bed, and the steam conducted underneath the blankets. If dry hot air is used, this can be conducted underneath the bedclothes by means of a small stovepipe about three inches in diameter—two short lengths with an elbow. To the lower end of the pipe a small hood may be riveted, in which the alcohol lamp is placed. The portion of the pipe extending under the bedding should be covered with asbestos to prevent burning the patient or bedding. Here, as with the steam, a frame should be placed about the patient to support the blankets, so as to permit of free circulation of the air. A small portable cabinet may be used, but it is not as safe or comfortable for the patient as a sweat given with the body in a horizontal position. Brieger and Diesselhorst claim to have obtained a more concentrated perspiration, that is, containing more solid waste, when they used electric lights for generating the heat.

Some patients will sweat very satisfactorily if placed in a hot bath, then put to bed and covered up well with blankets. Pilocarpin, on account of its depressing effect, is rarely desirable.

Summary.—Sweats are indicated in patients with marked renal edema, threatened uremia, or where extreme hypertension exists. The amount of water and solid constituents eliminated in a sweat may be sufficient to lessen an edema or alleviate a threatened uremia. The manner in which the sweat is given is of little importance. The only points to be considered are its efficacy and the comfort of the patient.

Diet.—Diet is one of the most interesting problems in the therapy of nephritis. Formerly the strict milk diet was considered the only safe one. With increased knowledge of the functioning power of the diseased kidney, a more liberal diet is permitted.

1. See that the patient receives sufficient food, and that it contains at least the minimum amount of the various food principles.
2. Avoid all the foods or drinks that contain substances which, in the course of elimination, irritate the kidney.
3. Avoid an excess of those foods the waste of which is eliminated chiefly through the kidney, for instance, the proteins. In considering this point it will be necessary to determine in a general way the ability of the kidney in nephritis to eliminate nitrogen. This can best be accomplished by determining the blood urea nitrogen.
4. Avoid, at least in certain cases, foods containing large amounts of sodium chlorid.

Metabolism proceeds in nephritis in the same manner as it does in the normal individual, and the rules governing the required amount of the various food principles are the same. There is no ground for believing that a nephritic can get along safely upon a smaller amount of albumin than the healthy person. If not given the requisite quantity, he draws upon the body albumin. It is true, for a short period of time, the amount of albumin may be restricted; and if it is determined that the ingestion of proteins leads to retention of nitrogen, then he should be restricted to the minimum amount of albumin that is necessary to protect him from using up his muscle protein. The amount of albumin in the urine varies greatly from day to day, even on a fixed diet; and considerable care must be exercised in drawing conclusions from the degree of albuminuria. Voight's figures have been generally accepted, that the minimum daily consumption of protein should not fall below 85 gm. Chittenden considers these figures far too high, and has demonstrated that a working man may live on 40 gm. of protein daily. Taking 50 gm. as the minimum daily requirement, this would be contained in the following amounts of the various foods in the fresh uncooked state:

Hen eggs	7	
Milk	1,660 c.c.	1½ qt.
Lean beef	220 gm.	7⅓ oz.
Ox tongue	300 gm.	10 oz.
Lean veal	260 gm.	8⅔ oz.
Calves' liver	260 gm.	8⅔ oz.
Lean mutton	250 gm.	8⅓ oz.
Lean pork	240 gm.	8 oz.
Very fat bacon	3,000 gm.	100 oz.
Chicken breast	250 gm.	8⅓ oz.
Pigeon	210 gm.	7 oz.
Goose	320 gm.	10⅔ oz.
Rabbit	200 gm.	6⅔ oz.
Venison	230 gm.	7⅔ oz.
Trout, pike, herring	260 gm.	8⅔ oz.
Salmon	230 gm.	7⅔ oz.
Lobster	300 gm.	10 oz.
Oysters	800 gm.	26 oz.

COOKED BOILED MEATS

Lean beef	150 gm.	5 oz.	Lean pork	175 gm.	5½ oz.
Lean veal	190 gm.	6⅓ oz.	Chicken breast ...	160 gm.	5⅓ oz.
Lean mutton	160 gm.	5⅓ oz.	Salmon	210 gm.	7 oz.

BAKED OR FRIED MEATS

Roast beef	200 gm.	6⅔ oz.	Roast veal	200 gm.	6⅔ oz.
Beefsteak	200 gm.	6⅔ oz.	Lamb chop	220 gm.	7⅓ oz.
Stewed beef	160 gm.	5⅓ oz.			

The individual of average weight will require, if at rest, about 2,200 calories daily. If working, this will be increased to 2,800 or 3,000 calories. If the patient is confined to the minimum amount of albumin—50 gm.—he obtains from this source about 200 calories. The remaining 2,000 or more calories he takes in the form of carbohydrates and fats, and these must be taken abundantly. As the pure fats and sugar do not contain any protein, these may be given very freely. Each gram of sugar is equal to 4 calories. This the patient may take in the form of sugar or in jellies. A patient, provided it does not excite digestive disturbances, may take daily, in one form or another, 250 gm. of sugar, equal to 1,000 calories. The fats may be given in the form of butter and cream, and if the patient is taking meats, in the form of fat meats or oil on salads. One-half liter (1 pint) of 20 per cent cream is equal to 1,080 calories, while the same amount of milk is equal to only 325 calories. Cream contains slightly more albumin than milk, but on account of its high caloric value, a patient may be properly nourished with it and still receive the minimum albumin requirement. A patient on a pure milk diet, in order to obtain 2,200 calories, must drink 3.4 liters, or about 3½ quarts. In this amount he would receive 102 gm. of albumin, or more than twice the minimum requirement. If he takes 20 per cent cream, only 1 liter (1 quart) would be necessary to give him 2,200 calories, and this would contain less than 40 gm. of albumin. For this reason cream is much more suitable in nephritis than milk. The cream may be given in the form of cream soups, with cereals or bread, or made into a palatable drink by diluting with equal parts of seltzer or Vichy.

The various cereals contain from 7 to 12 per cent of protein. Peas and beans, 23 to 25 per cent. The various breads from 6 to 8 per cent, puddings from 3 to 6 per cent, soups ½ to 2 per cent, with the exception of pea, bean, and cream soups, which have 3 to 4 per cent. Green vegetables contain from 2.7 to 3.5 per cent. The root vegetables rarely contain more than 2 per cent of albumin. The fruits contain from 0.3 to 2 per cent of albumin. Most of them less than 1 per cent. Nuts vary considerably in the amount of albumin: almonds, 34 per cent; chestnuts, 10.7 per cent; walnuts, 15.7 per cent; hazel nuts, 17.4 per cent. Cream cheese contains only 2.8 per cent of albumin, but Stilton, Camembert, Swiss cheese, Edam, Roquefort, and Holland cheese contain from 22 to 29 per cent.

Extreme restriction of the protein is only advisable in the acute stage of nephritis. As soon as recovery is well advanced, this amount should be increased to 50 or 60 gm., or not to exceed 1 gm. per kg. of body weight. In chronic nephritis marked restriction of protein is only required in those patients who show nitrogen retention. The most scientific method of dieting these patients is to determine the blood urea and be guided by these findings. Protein may temporarily be restricted well below the physiological requirement.

The Form in Which the Protein Is Taken.—The belief has been prevalent that the diet in acute nephritis should consist chiefly of milk. This practice is not based on its digestibility, but with a view of lessening the work of the kidney. As a quart of milk contains 30 gm. of protein, in order to keep the patient properly nourished an excess of protein must be given, and thus the kidney is overtaxed. As the protein restriction is the important factor, is there any objection to giving it in the form of egg, meat, and vegetable albumin? This is not merely a question of theoretical interest, but of practical value, as the patient on a pure milk diet, if properly nourished, must take at least 3 liters, giving thus an excessive amount of protein. In addition, some patients object to milk, and most of them soon tire of it as an exclusive diet. Considerable work has already been carried out on this phase of the subject, and the results are quite uniform. The chief difference in the form in which nitrogen is eliminated on a meat or a milk diet is that in the former more nitrogen appears in the form of nitrogenous extractives. Whether white meats contain less of these extractives than dark meats has not been determined. Although considerable work has been done upon these subjects, the results are so conflicting that it is impossible to draw any definite conclusions. It is, however, probable that the amount of extractives in light and dark meat is practically the same, as it is only on this basis we can explain the conflicting results. Kaufman and Mohr have studied the effect of light and dark meats on the nitrogen elimination, albuminuria and clinical course of chronic parenchymatous and chronic interstitial nephritis. They conclude that it is a matter of indifference whether white or dark meat is taken. These results have been confirmed by von Noorden, Pabst, and others. The teaching that white meat is preferable to dark meat in nephritis is so thoroughly disseminated that it cannot be uprooted at once. However, increasing evidence is being collected that this view is fallacious.

Another interesting point is the effect of meats on blood-pressure. Dale and Dixon and Barger and Walpole have found that decomposing meat contains a pressure-raising substance belonging to the amino group.

These pressor substances may also be formed by the action of intestinal bacteria on tyrosin. It is possible changes of this character may take place in the intestines of man, and there is some clinical evidence that a person partaking freely of meat has a higher blood-pressure than when on a meat-free diet. It has been demonstrated that in nephritis the giving of large amounts of protein may increase the blood urea. This is without effect on blood-pressure. In other words, the blood-pressure is not apparently dependent upon nitrogen retention.[1]

There can be no objection to albumin in the form of eggs, as they are free from the extractives which are found in meats. To avoid alimentary albuminuria, the eggs should be cooked. Hen eggs contain about 7 gm. of albumin.

Foods directly irritating to the kidney should be avoided. This is especially important in the acute forms of nephritis. Our actual knowledge of this subject is very meager, but, following custom, certain foods or condiments may be considered harmful. In this list may be included the meat extractives which are present in all meat soups; and their use in acute nephritis is harmful as, in addition to the extractives, they contain large amounts of sodium chlorid, have little nutritional value, and increase materially the fluid intake. Condiments, as pepper, mustard, horseradish, and the various other meat sauces, are considered renal irritants, as are also certain vegetables, as radishes and onions. Inasmuch as our knowledge of this subject is very incomplete these precautions may be quite unnecessary. There is no objection to vinegar and lemon juice.

Coffee, Tea, Tobacco.—There is no good reason why coffee and tea in moderate amounts may not be taken, even by patients with acute nephritis. The former view, that caffein caused diuresis by stimulating the renal epithelium, has been disproved. Patients with nephritis are often needlessly restricted in their diet, especially in the withdrawal of tea, coffee, and tobacco. There is no reason to believe that smoking in moderation will have any effect upon the kidney. The effect of smoking on blood-pressure varies. In some individuals there is a fall; in others a moderate rise. It is well known that smoking will disturb the cardiac rhythm, especially when indulged in too freely. Coffee, tea, and tobacco are objectionable when used to excess, and to avoid this it may be advisable to forbid their use entirely.

Alcohol.—Alcohol rather rapidly passes into the tissues from the blood, and is there slowly oxidized. Traces of alcohol may appear in the blood for twenty-four hours after its ingestion. Small amounts of alcohol may be excreted unchanged through the kidney. There is still considerable difference of opinion in regard to the effect of alcohol on the kidney in acute nephritis; and the rôle it plays in the development of interstitial changes in the kidney. Until our knowledge of this subject is more definite, it is advisable to prohibit its use in all forms of nephritis. An exception may be made in the chronic alcoholics who develop nephritis, as this class of patients do not tolerate well the complete withdrawal of alcohol. When given it should be in a dilute form and in very moderate amounts.

---

[1] Epstein recommends the use of a high protein, low fat diet in certain types of nephritis, where the urine contains a large amount of albumin, as he claims that in these cases the serum protein is reduced. Confirmation of the good results from this method of treatment must be obtained before it should be extensively put into practice, on account of the danger of increasing the blood urea and thus precipitating uremia.

Summary.—The dietary treatment of nephritis consists:

1. In giving the patient the minimum amount of protein that can be allowed without leading to destruction of body protein.

2. In avoiding foods or drinks directly irritating to the kidney, as certain sharp vegetables, condiments, and alcohol.

3. In furnishing the patient with sufficient food to meet the body demands. For short periods of time the diet may be greatly restricted, as in uremia, with anuresis, 500 c.c. (1 pint) of cream daily. Although apparently it is a matter of indifference in what form the patient receives his protein, in acute nephritis the most satisfactory form is as milk and vegetable proteins. In all forms of nephritis there is no objection to the free use of fats and carbohydrates, and with the restriction of proteins these should be given freely. Sugar, jellies, cooked fruit, with plenty of sugar, have a high food value, and may be given in acute nephritis. Cream is much better than milk on account of its high caloric value, enabling a patient to receive sufficient nourishment without excessive albumin. There are many objections to the prolonged use of a pure milk diet. It furnishes too much fluid, too large an amount of albumin and an excessive amount of sodium chlorid.

In chronic nephritis without nitrogen retention there is no objection to the use of a moderate amount, 250 gm. (8 oz.) of meat daily. It is a matter of indifference whether this be light or dark meat (chicken or roast beef). The protein in chronic nephritis need not exceed 1 gm. per kg. of body weight, and may temporarily be reduced below this level. Alcohol is only permissible in those addicted to its use, and then in moderate amounts, well diluted. Coffee and tobacco, if properly restricted, probably do little harm.

Treatment of Hypertension.—The nature of the changes responsible for the high blood-pressure in nephritis has already been considered. In the pure parenchymatous type of nephritis in man the blood-pressure is rarely increased. It is especially in the vascular types, acute glomerular and chronic interstitial forms, that hypertension prevails. The increased blood-pressure may be considered an effort to pass more blood through the kidney, and thus increase elimination by this channel. Loeb's work on this subject is especially interesting, as he found that lowering the blood-pressure 35 to 45 points by nitroglycerin diminished the elimination of both water and solids. If further investigation supports this view, then lowering blood-pressure by drugs may be harmful rather than beneficial. In undertaking investigation on the effect of various agents in reducing hypertension, it must be borne in mind that the normal variation in pressure in this class of patients may be very great. When the pressure is taken daily for several weeks, fluctuations of 30 to 40 mm. are not uncommon. One patient studied by the author had for one week a blood-pressure of 200, then without apparent cause it fell to 110, where it remained for a period of two weeks. Following this and without any change in the treatment it rose to 220, and at this time the patient suffered from marked uremic symptoms. Such extreme fluctuations are extremely rare. Figure 1 shows the fluctuation in pressure extending over a year in a bedridden hemiplegic, with interstitial nephritis. Some of the high readings could be accounted for by worries, as the patient had family troubles which at times greatly excited her.

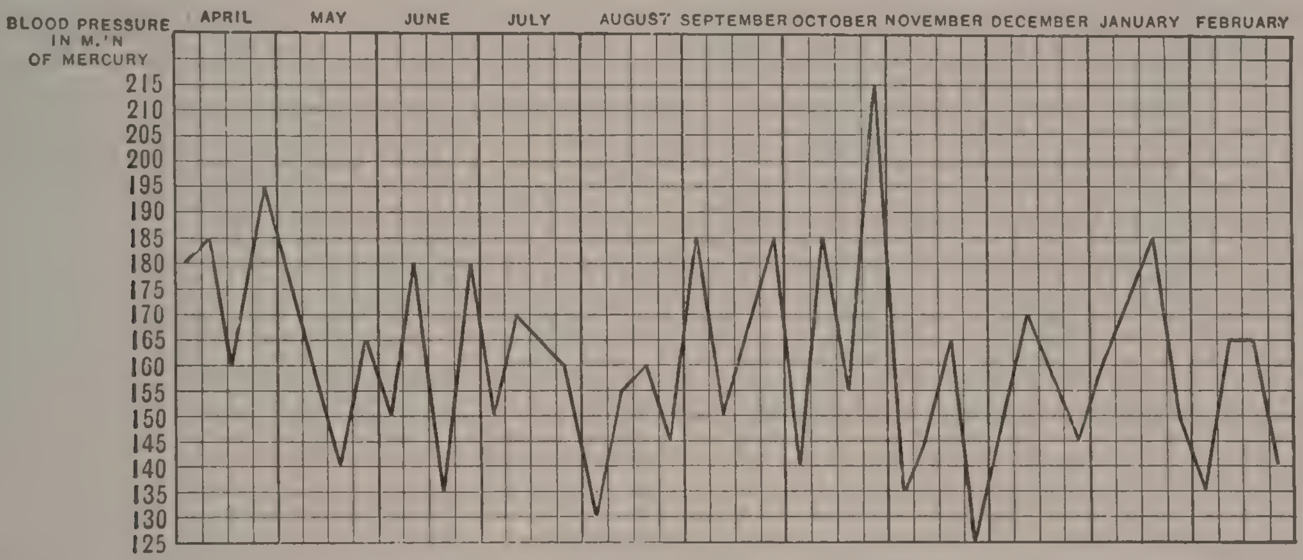


FIG. 1.—FLUCTUATION IN BLOOD-PRESSURE IN A PATIENT WITH HYPERTENSION.

Another point to be borne in mind when studying blood-pressure is that several readings, at an interval of perhaps a minute, should be taken before deciding what is normal pressure; as the excitement and pain due to the compression may produce a rather decided temporary rise.

Reduction by Increased Elimination.—Considering that hypertension in nephritis is due to retention of waste products, the most rational method of reducing it is to increase elimination through other channels, especially the bowels and skin. Free catharsis and gentle perspiration are safe measures, and may be employed even when the rise in pressure is only moderate. Even profuse sweating, if properly controlled, is a safe and efficient measure. Those foods, the waste products of which are eliminated through the kidney, should be restricted. This phase of the subject has already been considered under diet. In a series of cases the author has observed the effect of profuse sweats of twenty to thirty minutes on the blood-pressure. The reduction varied from ten to twenty points. The subjective relief obtained by the sweats is apparently largely independent of the changes in pressure. The redistribution of blood is probably an important factor, the dilatation of the cutaneous vessels relieving the congestion of the splanchnics. This change is analogous to what takes place in the Nauheim baths, in which the calcium and sodium salts and the carbonic acid gas, by their irritating action, produce distinct hyperemia of the skin. After a sweat, a period of three or four hours may elapse before the pressure returns to the previous level. When daily sweats are given for a period of two weeks or more, a patient occasionally shows a temporary fall in pressure, it remaining below the original level even for a few weeks after the baths have been discontinued. One patient, where the pressure was taken at irregular intervals for three years, who had never been below 210, after five sweats remained for ten days below 185 mm. In this case, with the reduction in pressure, the patient suffered from repeated attacks of transitory hemiparesis with numbness. As he previously had been entirely free from attacks of this character, and they entirely disappeared soon after the sweats were discontinued, it is probable that they were due to the sweating. This can best be explained by assuming that the brain could only be sufficiently supplied with blood through the narrowed vessels when the pressure was very high. Lowering of the pressure led to cerebral ischemia with transitory hemiparesis. Disturbances of this character have also been observed after the use of vasodilators.

Venesection.—Butterman bled 10 students, withdrawing, on an average, 300 c.c. (10 oz.). Nine had a fall in pressure varying from 5 to 30 mm. The author has noted the effect on blood-pressure in 5 cases after withdrawing 500 c.c. of blood. No effect was observed in 1 case, the other 4 showing a reduction varying from 5 to 15 mm. The effect upon pressure is dependent largely upon the rapidity with which the blood is withdrawn, slow bleeding often having very little effect. The results of venesection are not sufficiently striking to warrant its use except in those patients who are full-blooded. It would appear that much the same results, as far as reduction of pressure and removal of waste are concerned, can be accomplished by the sweating and without so severely taxing the patient.

Vasodilators.—Catharsis, sweating, bleeding, and rest may be considered rational measures for the reduction of pressure when due to retention of waste products. Reduction of pressure by the use of vasodilators is purely palliative, and, as has been mentioned, may do harm by lessening elimination through the kidney. Their use had best be restricted to those patients with threatened cardiac insufficiency, the result of hypertension, and to cases of extremely high blood-pressure, where there is danger of hemorrhage from vascular injury. Sodium nitrite and nitroglycerin are the two vasodilators most frequently employed. Erythrol tetranitrate has been less extensively used.

Nitroglycerin is best administered in the form of a 1 per cent alcoholic solution. This preparation is much more reliable than the tablet form. There is considerable difference of opinion in regard to the dosage. Enormous doses, 0.3 gm. (5 gr.), Stewart, von Noorden; 0.5 gm. (7½ gr.), Hochbauss, have been given without ill effect. On the other hand, severe collapse has occurred after comparatively small doses, 0.5 mg. (1/120 gr.), Loeb. There is evidently variation in the tolerance of different individuals. For the initial dose, 0.6 mg. (1/100 gr.) is sufficient; if there is no effect on blood-pressure within ten minutes, a second larger dose may be given. It can be frequently demonstrated that those patients who fail to react to moderate doses, 1 mg. (1/60 gr.), rarely show a fall in pressure after larger amounts. When given under the tongue, the fall in pressure begins within two minutes, reaches its maximum in five to ten minutes, then the pressure gradually rises, until within thirty-five to forty minutes it has almost reached the original level. Nitroglycerin, when given to patients with extreme hypertension, does not always lower blood-pressure. In a series of 10 cases only 7 reacted by a fall in pressure. In those susceptible to it the amount of fall in pressure varies greatly—20 to 50 mm. is about the average.

Sodium nitrite, on account of its prolonged action and failure to produce headache, is the most satisfactory vasodilator. The usual dose is 0.12 gm. (2 gr.). When given in solution the effect appears within five minutes, reaches its maximum in from eight to fifteen minutes. The return to normal is more gradual than with nitroglycerin. Like nitroglycerin, not every patient with hypertension is affected by it. The average fall in pressure is about the same as with nitroglycerin.

Erythrol tetranitrate in 60-mg. (1-gr.) doses produces a more marked reduction of the blood-pressure than either nitroglycerin or sodium nitrate. After 1 gr. a patient of the author's reacted by a fall in pressure of 110 mm. The first effect appears within four minutes, reaches its maximum in about ten minutes, and this maximum effect is more prolonged than either of the foregoing. As with other vasodilators, about 30 per cent of patients with hypertension fail to react. The chief objections to its use are its cost and the frequency with which it excites intense headaches.

The chart on page 544 shows the comparative effect of the various dilators on a series of ten patients.

Under the name of vasotonin, Müller has prepared a combination of yohimbin—nitrate and urethan. This depresses blood-pressure by dilating the peripheral vessels. Fellner reports that, when given subcutaneously, in man it reduces blood-pressure without affecting the heart. The dose, 10 mg. (⅙ gr.), is given every second day. The preparation is ex-

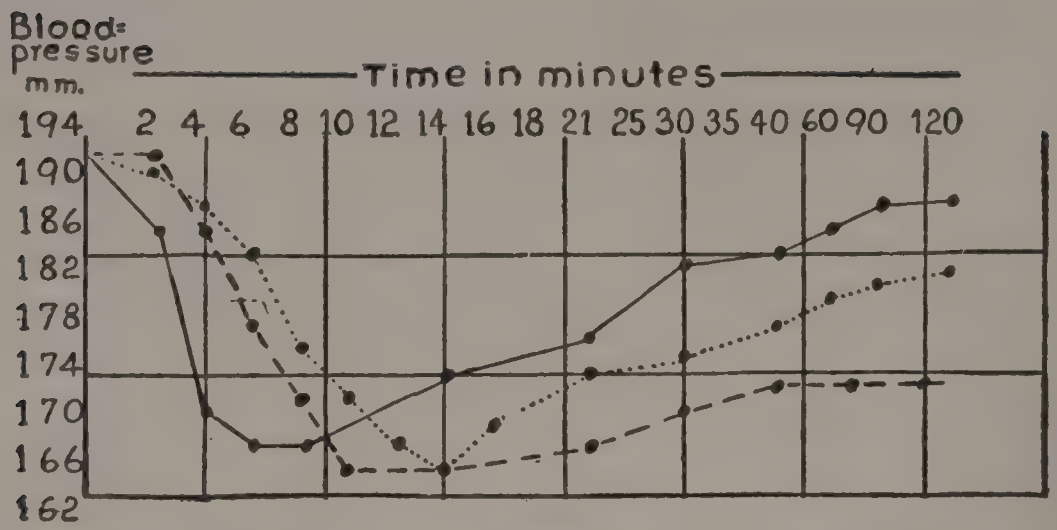


FIG. 2.—COMPARATIVE EFFECT ON BLOOD-PRESSURE OF NITROGLYCERIN (SOLID LINE), ERYTHROL TETRANITRATE (LINE COMPOSED OF DASHES) AND SODIUM NITRITE (DOTTED LINE).

pensive, and more extensive clinical reports are necessary before definite statements can be made in regard to its value.

In the treatment of the headaches associated with high blood-pressure the vasodilators are of little value. The various coal-tar products often give considerable relief, and in moderate amounts may be resorted to in case the pain is severe. High frequency electricity has been recommended for the reduction of blood-pressure. Arnold reports his results in 270 cases where the average fall in pressure was 12 mm. With the wide normal variations in pressure the question arises whether this change was really due to the treatment. The author has never seen any lasting results from its use.[2]

Summary.—Hypertension in nephritis is probably a compensatory process and should be treated by attempting to remove the cause. With our present knowledge, it appears that imperfect elimination through the kidney may be an important element in its development. If this is true, the rational treatment is to restrict those foods the end-products of which are eliminated through the kidney, for instance, proteins; to increase elimination through the bowel and skin by free catharsis and sweats. Vasodilators relieve the condition temporarily without removing the cause and are chiefly indicated, and then in conjunction with the above-named measures, when the pressure becomes exceedingly high or on the appearance of evidence of cardiac incompensation. In many cases of extreme hypertension the ordinary vasodilator drugs fail to reduce pressure. In all cases the reduction in pressure is transitory in character. Sodium nitrite, on account of its more gradual effect, prolonged action, and absence of untoward symptoms, especially headaches, is the most satisfactory drug of this group.

Use of Diuretics.—Diuretics may be used in all forms of nephritis, provided we avoid those preparations which act as intense kidney irritants. Turpentine, cantharidin, juniper berries, and gin should be avoided not only in the acute, but also in the chronic, forms of nephritis. The rather common practice of the lay people to administer gin in cases of anuresis is distinctly dangerous.

---

[2] The Editor wishes to confirm these negative results.—Editor.

The various alkaline diuretics, as the citrate, acetate, and tartrates of sodium, potassium, and ammonia may be given. Smillie has shown that in nephritis sodium salts are preferable to potassium. The nitrates may also be used, but are somewhat more irritating to the kidney. In nephritis with edema, however, alkaline diuretics usually fail to produce any diuretic action. The acetate of ammonia, sodium, or potassium is probably most frequently prescribed. They may be given in gram (15 gr.) doses in solution. Liquor ammonii acetatis, 10 to 25 c.c. (2½ to 6 fluidrams), or liquor ferri et ammonii acetatis, 15 to 30 c.c. (3 to 7½ fluidrams) are favorite preparations, the latter especially, as its iron content may assist in combating the anemia. It is true that the ammonia is oxidized in the tissues and excreted as urea, thus increasing the work of the kidney. However, the amount is so small that it can be overlooked.

Caffein or theobromin-sodiosalicylate (diuretin), and theocin, an artificial theophyllin, are more powerful diuretics than the alkaline group. In addition to their effect on the kidney, they stimulate the heart, and thus assist in causing diuresis. Caffein was formerly thought to excite diuresis by direct stimulation of the renal epithelia. It has been demonstrated by Loewi and others that the diuresis is due to dilatation of the renal vessels and interference with reabsorption of water in the renal tubule. Christian and others have shown that apparently in acute experimental nephritis theocin is injurious to the kidney. However, it may be tried out carefully; in case it excites marked diuresis it may be continued, but if without result it should of course be discontinued. Farr and Walker have shown that theocin, while increasing the water and salt output, does not increase the nitrogen excretion. In some patients with chronic nephritis and edema of renal origin theocin may excite marked diuresis as in a patient of the author's who lost 21 pounds in weight in six days. The ordinary dose of caffein is 0.15 gm. (2 gr.). Better results may often be obtained by intramuscular injection of some soluble salt of caffein, as the salicylate or sodium benzoate. In place of the caffein, diuretin or theocin, the former in doses of 0.5 to 1 gm. (7 to 15 gr.), the theocin in doses of 0.2 to 0.3 gm. (3 to 5 gr.). The diuretin is soluble and is best given in solution. The theocin is quite insoluble, and may be given in powder or capsules. Theocin sodium acetate is soluble and may be given, if desired, per rectum in 0.5 to 0.7 gm. (7 to 10 gr.) or intravenously in 5 gr. doses. A very serious objection to all caffein preparations is their irritating action on the stomach and their tendency to produce insomnia. The gastric disturbance may be avoided by giving the caffein hypodermically or in the form of rectal suppositories. As many of these patients already have difficulty in obtaining sufficient sleep on account of dyspnea or other causes, the additional insomnia caused by the caffein is a serious objection. Moreover, in severe forms of nephritis, with scant urine, caffein, like all other diuretics, usually fails. This restricts its field of usefulness, and it is only rarely, in cases where anuria is threatening, that caffein can be relied upon.[3]

Digitalis is another very valuable diuretic, and this action may be independent of its effect on the heart, as it has been shown by Jonescu and Loewi that the cause of the diuresis is a dilatation of the renal vessels due to a peripheral action and not to its cardiac effect. One objection urged against digitalis in nephritis is that it raises blood-pressure. Cushny, in a recent Harvey lecture, calls attention to this fallacy, as in man it does not cause a constriction of the peripheral vessels or a rise in blood-pressure. Indeed, in some cases it is distinctly lowered. This is due, according to Cushny, to the more perfect vasoconstrictor mechanism in man. With the increased systolic output following the use of digitalis, the vasomotor center receiving more blood, the tendency to increased blood-pressure is overcome by a compensatory dilatation of the splanchnic vessels. Even after intravenous use of strophanthin the rise in blood-pressure is negligible unless the individual's blood-pressure before the administration is high (Vagt). Eychmuller has shown that this is also true after the intravenous administration of digalen. There is no reason why a patient with evidence of cardiac incompensation due to high blood-pressure should not be given digitalis, and clinical experience has shown that the results are often very satisfactory. It is a matter of indifference in what form the digitalis is given. The superior claims made for the infusion are chiefly due to the relatively larger dosage of this preparation.

There is no evidence that the diuretics mentioned, except possibly the caffein group, are harmful. The great difficulty is that in cases of renal edema they usually fail to excite diuresis. When a diuretic fails to act after the lapse of a few days it is advisable to discontinue its use.

In the study of experimental nephritis some very interesting information has been acquired in regard to the effect of diuretics in acute nephritis. By employing different agents it is possible to produce in animals changes which are confined, early at least, chiefly either to the epithelium of the tubules, or the vascular apparatus, especially the glomerulus. Animals with the tubular type of nephritis respond to the action of diuretics. Later only, as the process extends and involves the vascular apparatus, do diuretics fail to act. Hedinger has shown that, in the early stages of this type of nephritis, caffein and digitalis give rise to unusually profuse diuresis. With the vascular type of experimental nephritis, from the beginning, diuretics are without effect, and no dilatation of the renal vessels can be demonstrated after their administration. This failure of response is coincident with the appearance of the edema, that is, where the kidney in nephritis reaches the point where it is unable to eliminate water, it also fails to respond to diuretics. This is in accord with clinical experience, that diuretics often fail to relieve renal edema. More important in the treatment of edema are the restriction of sodium chlorid and fluids and increased elimination through the bowel and skin. Inasmuch as acidosis may be marked in severe nephritis, due especially to the retention of phosphates, alkalis may be used to combat this. For this purpose bicarbonate of soda is most frequently used in quantities sufficient to produce a slight alkaline reaction of the urine.

---

[3] It is frequently satisfactory to start diuresis with one or two doses of theocin and to keep it up with diuretin.—Editor.

Summary.—The various alkaline diuretics, caffein, and digitalis may be safely administered in all forms of nephritis. Those diuretics directly irritating to the kidney, as turpentine, cantharidin, and gin, should be avoided. Animal experimentation and clinical experience have shown that when the kidney has lost its ability to eliminate water it has also lost its power to respond to diuretics.

Treatment of Uremia.—The rational treatment of uremia would be to increase the functioning power of the kidney. Unfortunately we have no means of accomplishing this and, therefore, our efforts should be directed toward restricting to a minimum the amount of waste to be eliminated through the kidney. This is accomplished by placing the patient upon a low protein diet, and in case there is evidence of poor elimination of water to restrict fluids. The second important measure is to increase elimination through other channels, for instance, bowel and skin. In acute uremia the restriction of proteins may be carried to an extreme degree. With chronic uremia such restriction is inadvisable. A certain amount of nitrogen—6 to 8 gm., equal to 35 to 50 gm. protein—will be present in the urine, even in starvation. In a protein-free diet this nitrogen arises from the destruction of body albumin. Lusk calls attention to the sparing influence of carbohydrates on protein metabolism. On an abundant carbohydrate diet, containing only 1 gm. of nitrogen daily, Landgren was able to obtain a urinary nitrogen of only 4 gm., while on a starvation diet more than double this amount of nitrogen appeared in the urine. Fats protect metabolism to a less degree, although on a diet containing half its calories in carbohydrates and half in fats the protecting power to the protein is the same as on a pure carbohydrate diet (Landgren). This shows the importance of giving a patient on a low protein diet an abundance of carbohydrates and fats, and especially the former. Fifty grams of protein daily is probably the minimum amount upon which a patient can be kept for a long period. Restriction beyond this point, except for short intervals, is not advisable. In the acute uremias, the proteins should be given in the form of milk, cream, cereals, or other carbohydrates with low protein content. The free use of carbohydrates is not objectionable and should replace the pure milk diet formerly recommended.

If the patient has marked edema, it is advisable to limit the daily fluid intake to one quart or less daily. Where there is anuresis with very little edema an effort may be made to excite diuresis by free water drinking, subcutaneous transfusion with normal salt, or with normal salt solution by rectum. If the kidney reacts to the increased fluid intake, it should be continued. Where diuresis does not follow such measures, and this is the rule, they should not be continued, as they will only increase the edema. Sugar may be given freely, provided the patient will take it, in the form of sweet lemonade, jellies, honey, etc. Cereals and bread are harmless, and these may be used as a vehicle for the cream. In fact, any of the carbohydrates are allowable. As a rule, however, in acute uremia the patient's appetite is much impaired, or he is comatose, so that it becomes very difficult to give food. For this reason it is usually necessary to restrict his diet to liquids, and for this purpose cream with cereals is most suitable. If improvement occurs, carbohydrates, as bread, sugar, jellies, etc., may be given in order to increase the caloric value of the diet.

Patients with threatened uremia should be placed upon a low protein diet with plenty of carbohydrates. One pint of 20 per cent cream (equal to 1,000 calories) contains about 18 gm. of protein. One quart of milk (equal to 650 calories) contains about 30 gm. of protein. This shows the advantage of the cream over the milk. In addition, the patient may receive various carbohydrates, bread, cereals, cooked fruits, jellies, etc. When necessary, these foods may be used as a vehicle for the cream. The quantity of cream may be increased if necessary to 1½, or even 2, pints (1,000 c.c.) without increasing the amount of protein beyond the permissible quantity. The fluid intake in threatened uremia depends upon the permeability of the kidney to water. If this is good, abundant water may be advantageously given. If edema is present, indicating inability of the kidney to secrete water, then the amount of fluid should be restricted. When fluids are indicated, there is nothing better than good plain water or fruit juices.

Free elimination through the bowels is desirable. For this purpose the saline cathartics are most commonly employed. Where the patient objects to these, compound jalap powder, elaterin, phenolphthalein, or any other laxative that will produce a watery movement may be used.

Elimination through the skin by means of vigorous sweats once or even twice a day is of great value and should be attempted in all cases of acute uremia. Rarely is a patient's heart so weak that he cannot safely take a good sweat, if properly administered and supervised. The method of giving the sweats has already been considered. Venesection may be of value, especially in well-nourished, full-blooded persons. It is questionable whether it has many advantages over sweating, still in urgent cases 500 to 750 c.c. (17 to 25 oz.) of blood may be withdrawn from one of the superficial veins by cutting down on the vein, or by introducing into it an aspirating needle, and allowing the blood to escape. If this last method is employed and the needle tends to become obstructed, the clot may be removed by using a little suction. If the patient is not edematous, following the venesection, he may be transfused with ½ liter of normal salt solution. Especially in comatose patients, venesection and transfusion may give decided relief.

The various diuretics, especially the caffein preparations and digitalis, may be tried. On account of the nausea and the tendency of caffein to irritate the stomach, it is best given hypodermically, using some soluble salts like caffein sodium salicylate, or benzoate, 0.15 to 0.2 gm. (2 to 3 gr.) every four hours. Digitalis is chiefly of value where cardiac impairment is present.

When the patient is edematous, drainage by incisions or puncture of the legs is indicated. The draining of such accumulations is always advisable, as it avoids the necessity of reabsorption into the blood of material which is supposedly toxic. The relief obtained by draining from the extremities often enables the patient to rest more comfortably and perhaps obtain some needed sleep. If the body cavities contain any considerable amount of fluid, it should be aspirated.

It is exceedingly difficult to relieve the headache of which many uremics complain. Increased elimination is most essential. Nitroglycerin or sodium nitrite rarely gives relief, and not infrequently increases the pain. Many of these patients with severe headaches find of their own accord that coal-tar products give considerable relief. They tend to lower blood-pressure, although it can be demonstrated that patients with hypertension who obtain relief from their headaches by taking coal-tar products frequently do not have a reduction in pressure when the pain disappears.

The uremic vomiting is rarely benefited by the use of bismuth or other gastric sedatives. If the patient's condition warrants it, lavage of the stomach frequently gives temporary relief. It is dangerous, however, to attempt to pass the stomach tube if the blood-pressure is very high or the patient's heart action poor. Drinking large amounts of water may wash out the stomach and give temporary relief. In many of these cases the vomiting is central in origin, and local measures are of little value. Where the vomiting is intense and persistent the question may arise as to the advisability of using morphin. The chief objection to its use is that it constipates and lessens the secretions from the skin, and also probably the secretion of urine. In spite of these objections there are occasions when relief from the vomiting becomes imperative on account of exhaustion and a hypodermic of morphin, 8 to 16 mg. (⅛ to ¼ gr.), may be given.

To control the extreme restlessness, 0.7 gm. (10 gr.) of chloral may be given by mouth, or in case the patient vomits 1 to 2 gm. (15 to 30 gr.)

per rectum. Morphin is not desirable, but may be given if other measures fail to control the restlessness.

Uremic Dyspnea and Pulmonary Edema.—The dyspnea associated with renal diseases has usually been considered of cardiac origin, but recently it has been shown that increased carbon dioxid tension due to the acidosis may also be a factor. With this in mind the alkalis may be used freely, as sodium bicarbonate 5 to 10 gm. (75 to 150 gr.) daily or until the urine becomes slightly alkaline. Rarely is there a real bronchial asthma. So-called uremic dyspnea is almost invariably associated with hypertension and not infrequently passes into pulmonary edema, as both are due to the same causative factors. As a result of the high blood-pressure, the left ventricle is unable completely to empty during systole. When this residual blood has accumulated until there is insufficient room in the left ventricle for the volume received with each systole from the right heart, there follows hyperemia of the pulmonary vein and artery, the lung becomes more voluminous, and its elasticity is impaired. Proper aëration of the blood is interfered with, partly from lessened flow of blood through the lung, and partly from the hyperemia with lessening of the air space. If this pulmonary stasis is not relieved, the pressure in the pulmonary artery increases and, with this, pulmonary edema develops. High blood-pressure, however, is not the only cause of acute pulmonary edema. This same disproportion between the working power of the two sides of the heart may arise where there is a weak left ventricle, without high blood-pressure; dilatation and stasis then occur. It has been the author's experience, however, that acute pulmonary edema in nephritis is confined chiefly to those patients with hypertension. The origin of uremic dyspnea and pulmonary edema has been given in some detail, as it gives us a clew to the method of treatment.

When associated with high blood-pressure an effort should be made to relieve the left heart. This may be accomplished by a vasodilator or much better by bleeding. Second, to stimulate the heart, and this can best be done by intravenous injections of digitalis or strophanthin. When experimental acute pulmonary edema has been produced in an animal by using adrenalin, the best results are obtained by bleeding from an artery, and at the same time injecting digitalis intravenously. By this combination the balance of the circulation is restored. When the pulmonary edema is due primarily to weakness of the left ventricle, without high pressure, venesection, with cardiac stimulation, fulfills the indication. Where the edema is associated with high blood-pressure bleeding gives better results than the vasodilators. In all cases of acute pulmonary edema in man morphin is indicated. Relief is obtained by its use more frequently than by other means. Its method of action is not clear. Perhaps part of the benefit is due to allaying the patient's fear. Atropin has been recommended and, apparently in some cases, gives relief. Lead acetate, at one time highly recommended, is now rarely used. Adrenalin has been recommended in the treatment of acute pulmonary edema. In all cases, if given in a manner that is followed by its physiological effect, for instance, intravenously or subcutaneously, adrenalin is contra-indicated. Plenty of fresh air apparently is as beneficial as oxygen inhalation. To prevent attacks of acute pulmonary edema, the patient should avoid overexertion. Not infrequently such attacks are due to overtaxation, either physical or mental. Sweats and good elimination through the bowel are also of value in warding off attacks. For the Cheyne-Stokes' breathing, which so frequently interferes with sleep, small doses of opium may be of value; large doses are apt to make the condition worse. Relief is often obtained by the use of caffein, or by digifolin intravenously.

Summary.—So-called uremic dyspnea and acute pulmonary edema are different stages of the same process. To avoid overtaxation, physical and mental, and to maintain good elimination are the best prophylactic measures. During an attack of acute pulmonary edema with high arterial tension the indications are met by bleeding and at the same time slowly injecting intravenously some digitalis preparation, as digifolin. If it is thought inadvisable to bleed, use vasodilators, as nitroglycerin or sodium nitrate, and intravenous injections of digitalis. When the arterial tension is low, and there is evidence of venous stasis, perform venesection and intravenous injection of a suitable digitalis preparation. Morphin in moderate doses, 10 to 15 mg. (1/6 to 1/4 gr.) hypodermically is safe, and often gives decided relief. Atropin may be tried but in the writer's experience is of limited value. It is well to remember that attacks of acute pulmonary edema disappear, as a rule, within one or two hours even when untreated.

Attacks of acute pulmonary edema very frequently develop just after retiring. To ward off these attacks the patient should undress slowly, have the room and bed comfortably warm, and take, before undressing, 2 grains of sodium nitrite. If, in spite of these measures, the attacks persist, he should remain in bed for a week or more in order to rest the heart. To avoid attacks of acute pulmonary edema rest is most essential.

SPECIAL TREATMENT

ACUTE NEPHRITIS [4]

In acute nephritis there are active inflammatory or degenerative changes in the vascular, interstitial, or epithelial structures in the kidney.

---

[4] The classification of nephritis as given in the previous edition is retained. The classification given by Christian is more up-to-date. The profession generally, however, are more familiar with the form as retained here and it is quite satisfactory from the standpoint of treatment.

While the most active change may be confined to one of these structures, all are usually more or less extensively involved, especially after the process has continued for some time. The treatment consists in prophylactic measures to prevent or modify the intensity of the inflammatory or degenerative changes, and active treatment of the fully developed nephritis.

Prophylaxis.—The chief etiological factors in acute nephritis have already been mentioned and briefly are acute or chronic infections, chemical agents, cold, and products of disturbed metabolism. Scarlet fever and diphtheria are the two acute infections most frequently complicated by nephritis. Chronic infections much less frequently cause trouble, although tuberculosis or syphilis are frequently responsible for a mild grade of acute nephritis. Acute nephritis not infrequently follows very mild acute infections, especially tonsillitis. Complete removal of the tonsils is advisable in children subject to repeated tonsillar infection, not merely to avoid nephritis, but to ward off attacks of acute articular rheumatism. Gargles and sprays are probably of little value as preventive measures, as the infection usually begins deep down in the crypts, far beyond the reach of local agents. Any tonsil, when associated with cervical glandular enlargement, should be removed.

It is questionable how much may be done in scarlet fever to prevent nephritis. The determining factors are probably the intensity of the infection and the individual susceptibility. At the present we do not possess any efficient measures for combating either the intensity or duration of the toxemia. Good elimination through the bowel, kidneys, and skin may assist in lessening the toxemia. This may be accomplished by carthartics, free fluid intake, and warmth. On account of the importance of cold in nephritis, the question arises as to the advisability of cold baths in the acute fevers, especially scarlatina. There is no positive evidence that patients so treated are more subject to nephritis. It is advisable, however, to avoid the very cold bath. Rest in bed during the acute infection, by maintaining uniform temperature of the skin and reducing body waste, lessens the amount of kidney work. The early recognition of diphtheria and prompt administration of antitoxin lessen the probability of nephritis.

In diseases that predispose to nephritis kidney irritants should be avoided. This includes salol, salicylates, potassium chlorate, mercury, especially bichlorid, turpentine, copaiba, cubebs, and cantharides. The condiments and sharp vegetables, like onions and radishes, have the reputation of being kidney irritants and, until their action has been definitely determined, should be avoided. Strong alcoholic drinks should not be used, as they undoubtedly irritate the kidney. Moderate amounts of alcohol probably are not injurious, but in those acute infections, which are prone to excite acute nephritis, it is safer to avoid alcohol entirely.

Active Treatment.—There is no specific therapy in nephritis. One's efforts should be directed, first, to limit extension of the process. Second, to put the minimum amount of work on the kidney by restriction of diet, and increased functioning of the other organs capable of assuming, in part, the work of the kidneys; third, the treatment of symptoms that arise, as edema, uremia, etc. Known kidney irritants in the form of foods should be avoided. Rest in bed is imperative, as it reduces to a minimum the body waste and offers the best protection against chilling of the skin. The body should be kept as warm as is consistent with the patient's comfort. Placed between warm woolen blankets, the patient should be allowed an abundance of fresh air. The mistake is frequently made of inducing heat by raising the room temperature to an uncomfortable degree. This is not only unnecessary but harmful. The skin should be thoroughly cleansed daily with soap and tepid water. Cold baths should be avoided. Cocoa butter and other oily preparations should not be applied to the skin, as they interfere with perspiration.

Catharsis.—The bowels should be kept free, the degree of catharsis depending upon the intensity of the intoxication and amount of edema. It is not advisable to resort to prolonged, extreme catharsis on account of the resulting emaciation. Laxatives producing a watery movement are most desirable. Magnesium or sodium sulphate in half-ounce (15-gm.) doses, in half a glass of hot water before breakfast, is most frequently employed and, when well borne by the patient, is entirely satisfactory. Sodium phosphate in half-ounce (15-gm.) doses in hot water is somewhat more palatable. Seidlitz powder may be preferred by some. The natural or prepared saline waters, provided they do not contain large amounts of sodium chlorid, make a rather agreeable form for administering the saline. Effervescing preparations are often more palatable—liquor magnesii citratis, 12 fl. oz. (360 c.c.), lithii citras effervescens, 1 to 2 dr. (4 to 8 gm.), magnesii sulphas effervescens, 1 to 4 dr. (4 to 16 gm.). When the stomach is disturbed calcined magnesia (magnesia oxidum), 5 to 60 gr. (0.3 to 4 gm.), is often well borne. Calomel is commonly used, but except for its slight diuretic action it does not possess any special advantages. Senna, cascara, rhubarb, aloin, elaterin, or any of the other vegetable cathartics may be tried. Elaterin, gr. 1/16 to 1/10 (4 to 6 mg.), gives a very watery movement, and is especially valuable in extensive edema, or threatened uremia. Another favorite laxative, although disagreeable to take, is compound jalap powder in dose of 15 to 60 gr. (1 to 4 gm.). When the patient is delirious or comatose, one or two drops of croton oil on sugar may be given. Enemata rarely give a sufficient movement, but may be advantageous when combined with laxatives.

Diuretics.—Diuretics may be tried to increase the flow of urine, but are rarely of value in acute nephritis. It has been shown experimentally that when a kidney fails to respond to simple diuretics like water it rarely responds to the more powerful ones. The acetates, citrates, or nitrates may be tried in doses of 15 gr. (1 gm.) dissolved in water. There is no objection to trying caffein, 2 gr. (0.12 gm.), theobromin sodium salicylate (diuretin) 10 to 15 gr. (0.7 to 1 gm.), theophylin 3 to 5 gr. (0.2 to 0.35 gm.), dimethyl xanthin (theocin) 3 to 5 gr. (0.2 to 0.35 gm.).

It was formerly urged that caffein preparations irritated the kidney and were contra-indicated in acute nephritis, but conclusive evidence that this is true when given in the ordinary therapeutic doses is still lacking. All caffein preparations are gastric irritants and tend to produce insomnia. For this reason their continued use is not advisable. A safe rule to follow in acute nephritis where diuretics are indicated is to determine the daily urinary output first, then try the various diuretics and determine if the output of water is increased. By following this method it can be decided whether in the individual case a diuretic effect is obtained, and thus avoid the useless administration of drugs. When the heart action is impaired digitalis often acts better than any other diuretic. Under its use the urinary output is increased and the edema subsides. It is largely a matter of indifference what form of digitalis is used, as the results are dependent upon the activity of the preparation. When we are sure of a fresh leaf the infusion is a very satisfactory preparation. In this country, however, where the average pharmacist pays little or no attention to the freshness of the leaves, it is better to use a tincture prepared by some reliable manufacturer. Digifolin is a reliable preparation. It is put on the market in tablet form, each tablet equivalent to 1½ gr. of the dried leaf, and also in ampules for intramuscular or intravenous use. High blood-pressure is not a contra-indication to the use of digitalis.

Use of Alkalis.—Recently Hosslin has shown that in albuminuria and in nephritis the amount of albumin in the urine can be lessened by the administration of alkalis, and in some cases the number of casts may also be reduced. Conzen has demonstrated that only the acute forms of nephritis are affected by this method of treatment. The amount of alkali required varies with the individual case, depending upon the degree of acidity of the urine. When possible the acidity of the urine should be determined and an alkali like bicarbonate of soda given in sufficient quantities to bring the urine close to the neutral point.

Diet.—The chief indication in acute nephritis is to avoid foods that irritate the kidney, that throw increased work upon the kidney, or are poorly eliminated through the kidney. When dieting is continued for more than a few days it is necessary to consider the caloric value of the food. Substances that irritate the kidney have already been considered. Orange or lemon juice is harmless and often of value in lessening the tendency to acidosis. There is no objection to moderate amounts of tea, coffee, or chocolate. On account of the usually poor elimination of urea the protein in the food should be reduced to the minimum consistent with protecting the body albumin. On a protein-free diet the body muscle is used, and a certain amount of nitrogen continues to be eliminated through the kidney. It is of advantage, therefore, to give sufficient protein to prevent muscle destruction. This amount may roughly be considered as 50 gm. or about the amount contained in a quart and one-third of milk. It can be readily seen that it is possible to administer excessive protein on a pure milk diet as the patient receiving three quarts daily would be taking about 120 gm. of protein daily, and to get rid of the consequent nitrogen waste must eliminate 30 to 35 gm. of urea daily. This far exceeds the ability of the acutely diseased kidney. Cream, on account of its high food value, is more desirable than milk. Easily digested carbohydrates should be used freely, and may make up a large part of the diet in acute nephritis. Where the edema is very marked fluids should be reduced to the minimum as judged by the patient's thirst, and carbohydrates substituted for milk on account of the salt content of the latter. Easily digested carbohydrates, as cereals, toast, light desserts, cooked fruits, fruit jellies; and some of the green vegetables, as string beans, asparagus, and spinach, furnish variety and add considerably to the food value of the diet. The addition of carbohydrates to the dietary is necessary when we wish to keep a patient for a prolonged period of time on a low protein diet as it protects the body protein. Abundance of sugar may be added to all the foods; in addition sweet lemonade, fruit jellies, honey, etc. Sugar has a high caloric value and is free from waste eliminated through the kidney. Cream further increases the food value of cereals and cooked fruits. Milk and cream may be given as cream soups, ice cream, milk chocolate, and in other forms to suit the patient's taste.

Creatinin, like urea, is not easily eliminated through the kidney. A considerable portion of the creatinin present in the urine arises from the destruction of body muscle. The chief source of ingested creatinin is meat extracts and meat broths. These should both be avoided in acute nephritis as, in addition to the creatinin, they contain rather large amounts of sodium chlorid and have a very low nutritional value.

Urinary pigments, phosphates, and hippuric acid are also eliminated with difficulty. The urinary pigments are derived almost entirely from the blood and, by omitting meat, we remove the main source of external supply. There is no means of reducing the amount of pigment derived from the normal destruction of red blood corpuscles. The chief source of phosphoric acid is meat, milk, yolk of eggs, and certain vegetables. In the ordinary diet of acute nephritis the chief supply comes from the milk. If we consider the amount of phosphates ingested in this way as harmful calcium carbonate may be given, as recommended by von Noorden. The calcium combines in the intestine with the phosphoric acid and renders it less readily soluble. Hippuric acid is formed in the kidney from benzoic acid. The amount of benzoic acid in the foods is practically negligible.

Sodium Chlorid.—Attention has been called to the importance of sodium chlorid restriction in nephritis. It is especially in the acute forms with edema that the beneficial results of its restriction are most marked. There is sufficient sodium chlorid in a quart of milk to supply the body requirement; another argument against the use of large quantities of milk in acute nephritis. No salt should be added to any of the food in acute nephritis with edema. The use of unsalted bread and butter may be advisable in extreme cases. Salted butter contains on the average 1 per cent of salt, and bread 0.5 to 0.7 per cent. As all the fruits and carbohydrates contain very small amounts of salt they may be used freely.

Fluid Intake.—Not every case of acute nephritis shows an inability to eliminate water. The presence of dropsy, however, is evidence of kidney impermeability to fluids, and taking large amounts of liquids only increases the edema. Where the kidney is permeable to water, as shown by the absence of edema, free drinking of liquids (and there is nothing better than plain water) increases the urinary output, not only of water, but also of solids; and flushes out the débris from the urinary tubules. It is scarcely necessary to state that the various waters recommended for the cure of nephritis do not possess any virtues over pure plain water; and some of them contain ingredients that are actually harmful to the kidney. Where the edema is marked the fluid intake should be restricted to the minimum; the patient's comfort being the only limitation. Each patient must be treated individually, being guided by the permeability of the kidney.

Sweating.—To increase elimination of solids and water, sweating is often desirable. The most marked benefit is noted in those patients with marked edema. It is possible with a good sweat to remove a quart of water and 15 gr. or more each of urea and sodium chlorid. In addition to the eliminated matter some benefit may be derived from the effect on the circulation, the peripheral vessels being dilated. Whether sweating ever increases the degree of intoxication, and also the manner of giving the sweat, have already been considered in detail under the general principles of treatment. Heat is the safest and most satisfactory diaphoretic.

Decapsulation.—Decapsulation, as introduced by Edebohls in 1901, has been largely abandoned. More careful clinical observations following the operation have failed to show that marked improvement resulted. Experimentally it has also been demonstrated that increased hyperemia of the kidney, as claimed by Edebohls, does not develop as a result of the operation. Although the operation is little resorted to at present, occasional reports show that in some severe cases improvement apparently follows, and it may be in a few instances the operation is beneficial. It is not, however, free from danger, and it is questionable whether it should ever be advised.

Edema.—The edema can best be controlled by restriction of fluids and salts, free catharsis, sweating, diuretics, and direct removal, by tapping or drainage. Where the edema is marked, the fluid may be safely restricted for a few days to 1½ pints, this consisting of equal parts of cream and milk. This also supplies the patient with a low sodium chlorid diet. Good results may often be obtained by the Karell cure, which consists in a marked restriction of both salt and liquids. For several days the patient receives 200 c.c. of milk at 8 A.M., 12 noon, 4 and 8 P.M., nothing else. After three or four days dry foods without salt may be added, but the liquids for a period of three to four weeks are restricted to 800 c.c. daily. Often during the first few days of the cure large amounts of sodium chlorid are eliminated and the beneficial results are due to the low salt and fluid ingestion. The subject of catharsis and sweating has already been considered. The alkaline diuretics, caffein preparations, or digitalis may be tried, but should be discontinued in case they fail to increase the urinary output. When fluid accumulates in considerable quantities in the body cavities, it should be withdrawn. When other measures have failed, incisions in the limbs or Southey's tubes may give great relief.

Anuresis.—Anuria is usually associated with edema. The kidney has temporarily become completely impermeable to water. Increasing the fluids rarely excites diuresis. Efforts must be made to increase elimination through other channels, until the kidney function is regained. Catharsis and sweating, combined with low protein diet and reduction of fluids, offer the best hope of relief. Severe anuresis is of short duration, the patient either dying or the urinary output increasing within a few days. Cream, cereals, bread, jellies, cooked fruits and other carbohydrates should constitute the diet. On account of the short duration of the anuresis, extreme restriction in diet may be resorted to.

Uremia.—The probability of uremia must be considered in every case of acute nephritis, and for this reason prophylactic measures should be instituted from the onset of the trouble. These measures have already been considered, for instance, low protein diet and good elimination through skin and bowel. When uremia has actually developed, these measures must be pushed to the limit. When the edema is marked, aspiration of the body cavities and drainage of the legs are indicated. By these measures considerable quantities of more or less toxic material are removed. Removal of fluid in this manner is to be preferred to bleeding, as such fluid contains practically the same amount of toxic material as the blood. Transfusion with normal salt is contra-indicated in uremia with edema. The kidney is impermeable to water and transfusion merely increases the dropsy. When uremia develops without edema transfusion may be of value. The normal salt may be introduced intravenously (1 quart) or subcutaneously in the same amount. If marked diuresis follows, the transfusion may be repeated every four hours until the intoxication is alleviated. When diuresis does not follow, it is inadvisable to repeat the transfusion. The introduction of normal salt per rectum by the drop method may be resorted to, only, however, in patients free from edema. As a rule, however, patients with uremia from acute nephritis have marked edema.

Dyspnea.—Dyspnea may be a very distressing symptom in acute nephritis. It is, however, much less frequent than in the terminal stages of the chronic interstitial type. It is most frequently of cardiac origin, although increased carbon dioxid tension due to the acidosis probably also plays a rôle, so the free use of alkalis as bicarbonate of soda may be of value. Digitalis may be tried. Withdrawal of fluid from the body cavities is often beneficial. Not infrequently a good sweat in the evening may give sufficient relief to enable the patient to obtain a night's sleep. When the dyspnea interferes with sleep, no drug gives as much relief as some form of opium. Although it constipates and lessens the secretion from the skin, it is nevertheless the safest remedy we can employ to induce sleep. With moderate doses, the secretion from the kidney is not affected.

Itching.—For the itching of the skin, so common in uremia, alkaline baths often give relief. We must be careful to avoid local applications that might irritate the kidney.

Backache.—Backache, which is quite common in acute nephritis, is often relieved by local heat—a hot-water bag, or, still better, an electric pad. Vigorous massage of the lumbar region may give considerable relief. Simple plasters at times are beneficial, probably largely due to retaining the heat.

Edema of the Glottis.—This condition may be treated by scarification of the edematous area and in severe cases tracheotomy may be necessary. Local cold to the neck may be safely tried. For the pulmonary edema morphin is the most satisfactory remedy. When the patient is not very anemic, and the blood-pressure high, withdrawal of a pint of blood may be beneficial.

Headache.—Local heat or cold may be used for the relief of headache. In case the heart action is good, some of the coal-tar products may be used. Headaches are usually symptoms of intoxication, and increased elimination is indicated.

Anemia.—For the anemia some simple form of iron, as the Blaud's pill, is satisfactory.

Gastric Disturbances.—These are uremic in origin, and local measures rarely give relief, although persistent lavage often is temporarily beneficial. Gastric sedatives are of little value. When nausea appears all drugs which might act as gastric irritants should be discontinued.

Hemorrhagic Nephritis.—Hemorrhagic nephritis does not require any special treatment. Ergot, suprenal extracts, and the various styptics do no good, and may derange the gastro-intestinal tract. Calcium lactate, gr. 15 (1 gm.), three times daily, may be used if the blood coagulability is lessened.

Summary.—In acute nephritis the patient should be placed upon a low protein diet, not to exceed 50 gm. daily. This amount is contained in 1½ quarts of milk. To increase the food value of the diet carbohydrates may be safely given. Cream is also valuable. When edema is present salt and fluids should be restricted. Good elimination through the skin and bowel is most important. Diuretics may be tried, but usually fail to act. Edema may be relieved by aspiration of the body cavities, or drainage from the legs by incision. When uremia is impending, elimination through skin and bowel should be augmented, and the protein content of the diet further restricted, by placing the patient upon 1 pint of cream daily, combined with simple carbohydrates.

Chronic Parenchymatous Nephritis

An acute nephritis may pass into a chronic form. More frequently, however, chronic parenchymatous nephritis begins as such. All of the etiological factors responsible for acute nephritis may also give rise to the chronic form. The more important of these are acute or chronic infections, lead, mercury, alcohol, chronic constipation, certain metabolic diseases, as gout and diabetes mellitus. The disease is more prevalent in cold or rather changeable climates, and chilling, no doubt, plays a rôle. The treatment of complications, as edema and uremia, has already been considered in the treatment of acute nephritis. The principles of treatment throughout are the same as those employed in the acute forms. The main differences are that here we are dealing with a chronic disease and the patient's method of living, as regards exercise and diet, must be so arranged as to be consistent with years of life. It is a disease characterized by repeated acute exacerbations, each of which leaves the kidney more impaired. During these attacks the treatment is the same as outlined in acute nephritis. Excessive muscular exertion, acute infections, chilling, errors in diet and intestinal intoxication are most frequently responsible for the acute exacerbations. Drugs that might irritate the kidney must be avoided. The degree of rest desired will depend upon the intensity of the process. Patients with slight uremic symptoms or marked edema belong in bed, or at least at rest. Patients with no evidence of the trouble, except the urinary findings, should be advised to avoid excessive muscular exertion and exposure to wet cold. It is advisable in even the very mild types to select a climate where the winters are mild. California is a very good all-year climate.

Elimination.—Good elimination through skin and bowel is important. Even the mild cases should take a daily warm bath. The degree of catharsis required depends upon the intensity of the process.

Diuretics.—Diuretics are more apt to be effectual here than in acute nephritis. When the edema is marked, caffein preparations or digitalis may be used. In the milder forms an alkaline diuretic, as liquor ferri et ammonii acetatis, ʒiv (16 c.c.), three times daily, possesses the additional advantage of supplying iron. As all the severe cases are decidedly anemic, iron in some form may be helpful.

Diet.—Diet is one of the essential parts of the treatment. The diet should be free of substances that might irritate the kidney, contain merely enough protein to supply the body needs, and possess the proper caloric value. The sharp vegetables, alcoholics, condiments, and meat soups containing considerable meat extractives, should be avoided. The amount of protein ingested is dependent partly upon the severity of the attack. The average patient can get along comfortably for an indefinite period of 60 gm. of protein daily.

Restriction of Fluids.—The restriction of fluids as recommended by von Noorden in certain cases of nephritis has been too generally applied. It is necessary to determine in every instance the permeability of the kidney to water. If this is good, then the patient should receive sufficient fluids to bring the output of the urine up to 1½ quarts in the twenty-four hours. In addition, once or twice a week, the patient may receive a flushing-out day when he receives fluids sufficient to yield 2 quarts or more of urine daily. This assures good elimination of nitrogenous waste. When the kidney is impermeable to water, as shown by the presence of marked edema, excessive water drinking only does harm. In these cases the bowel and skin must temporarily act vicariously for the kidney, as we cannot hope to eliminate the requisite amount of urinary waste through the natural channel.

Sodium Chlorid.—Comparatively simple rules can be established in regard to the use of table salt. Every healthy individual consumes many times the amount of sodium chlorid demanded by the body. This excess is eliminated largely through the kidney. In many cases of chronic parenchymatous nephritis the kidney is impermeable to salt. When taken in excess of the daily requirement it remains in the body and retains with it sufficient water to maintain the proper molecular concentration. This is the basis of Widal's theory of edema. It is advisable in every case of nephritis with edema to reduce the sodium chlorid to about 2 gm. daily. This is about the amount contained in 1 quart of milk. No salt should be added to any of the foods, except the small amount used in bread. Clear soup, which contains much salt, should be avoided. With every precaution to restrict salt, there is no danger of giving the patient an insufficient amount. Some advise carrying the restriction to the point of using unsalted butter. As the use of excessive amounts of salt is a habit, the patient soon becomes accustomed to the lessened quantity.

Tea, Coffee, Tobacco.—There is no objection, as far as the kidney condition is concerned, for the patient to use moderate amounts of tea or coffee. When insomnia exists the use of coffee should be restricted. Taken to excess, coffee acts as a cardiac irritant. Moderate smoking probably does little, if any, harm, as, with tea and coffee, it is the excessive use that is injurious. To deprive the patient with chronic parenchymatous nephritis of a cigar after each meal is, with our present knowledge of the action of tobacco, unwarranted.

Where we have a mixed form of nephritis, or when a chronic parenchymatous nephritis passes into a secondarily contracted kidney, the blood-pressure may be increased, and it becomes necessary to consider the proper treatment of the symptoms. This will be considered under the treatment of chronic interstitial nephritis.

Summary.—The treatment of chronic parenchymatous nephritis is in general the same as the treatment of acute nephritis. The chief point of difference is in the diet, as we are dealing with a chronic process and extreme restriction of protein is not feasible. Foods or drinks known to be renal irritants should be avoided. Carbohydrates and fats may be taken freely; the proteins can be safely restricted to 60 gm. daily. It is probably a matter of little importance in what form the protein is taken, whether in milk, meat, or eggs. In the presence of edema water drinking should be restricted. The salt should be restricted to that naturally present in the food. Edema is to be treated by restriction of fluids and salt, catharsis, sweats, diuretics, withdrawal of fluid from the body cavities, and, if necessary, incisions in the legs. Uremia is to be treated by good elimination and restriction of protein.

Chronic Interstitial Nephritis

The treatment of chronic interstitial nephritis is the same whether it be of the primary or secondary type. The only essential difference in the treatment and that of chronic parenchymatous nephritis is in the management of the high blood-pressure. Edema, when of renal origin, and uremic symptoms are treated in the same manner in all forms of nephritis. Probably every patient with chronic interstitial nephritis and compensated heart has an increased blood-pressure. As a rule, it is far above normal, not infrequently reaching 220 mm. of mercury. This throws an enormously increased amount of work upon the heart, which, sooner or later, may succumb to the strain. The main object of the treatment is, therefore, to lower blood-pressure and protect the heart. This increased blood-pressure frequently results in rupture of a blood vessel with fatal or serious hemorrhage. When a patient dies from the direct effects of a chronic interstitial nephritis, it is usually due to hemorrhage or cardiac incompensation. The treatment of the terminal stages of this form of nephritis is usually directed toward resting a broken compensation. Edema, when present, is, as a rule, of cardiac origin.

The theories regarding the cause of the hypertension have already been discussed. There is an increasing minority who look upon the hypertension as an effort toward physiological cure. With the impaired ability of the kidney to eliminate solids, the heightened blood-pressure causes increased blood flow through the kidney, increased elimination of water and conjointly of solids. The kidney has apparently lost its power to secrete a concentrated urine. The treatment should, therefore, be directed to the underlying cause, rather than directly at the hypertension. With our present knowledge, deficient elimination through the kidney is the apparent cause of the hypertension. Restriction of food excreted by the kidney and increased elimination through the skin and bowel is a more rational therapy than the use of vasodilator drugs.

Diet.—The protein diet should be restricted, as in the parenchymatous form, to 60 or 70 gm. daily. Where facilities are available for determining the blood urea, more or less than this amount may be given depending upon the absence or presence of urea retention. Nitrogen retention is far from constantly present in this form of nephritis.

The patient may partake freely of the green and root vegetables, cereals, fruits, etc. The only restriction necessary, as far as the kidney is concerned, is in the amount of protein.

It is quite important that the patient does not become obese. With the heart already overworked, a gain in weight is an added burden. To avoid increased weight, the sugars and fats may be restricted. On account of the danger of overtaxing the heart, severe physical exercise must be avoided, and the patient should depend mainly upon the diet for maintaining the proper weight. Restriction of fluids has been recommended by von Noorden, not on account of the fear of increasing edema, as these patients have good elimination of water, but because he believed excessive fluids increased the work of the heart. It is, however, impossible to raise the blood-pressure by the drinking of fluids to the point of satisfying thirst. Excessive reduction of fluid interferes with the proper elimination through the kidney. Probably the safest rule to follow is to gratify fully the patient's natural desire for water. When confined to water or milk, the patient will rarely take an excessive amount of fluids, unless urged to do so by his physician. Alcohol, if taken, should be limited to very small amounts. This may be permitted in elderly patients who have been accustomed to using it, and where its complete withdrawal may not be well borne.

Salt Restriction.—This is less essential in the interstitial type of nephritis than in the other forms, as renal edema is rarely marked. An excessive use of salt causes great thirst; its use may be objected to on this score. The ordinary foods supply sufficient salt, and it is unnecessary to add more. The average patient will soon become accustomed to taking his food with the addition, at most, of very small amounts of salt.

Hypertension.—Hypertension may be treated by increased elimination and by vasodilator drugs. The former method is the more rational. Vasodilators are used less and less frequently. The author has used them very infrequently in recent years, except in angina pectoris. Free catharsis and especially sweating, like all other methods, are transitory. When continued daily for two or three weeks, the pressure throughout the entire twenty-four hours is much reduced, only gradually returning to the previous level after the sweats have been entirely discontinued. By repeating such a course of sweats every three or four months, the patient may be more comfortable and his headaches and dyspnea less annoying. The sweating may be induced by hot wet blankets, hot bricks, or hot air in a special oven or cabinet. The duration of the sweat varies with behavior of the patient. When the respiration and pulse are good twenty minutes is a fair average. When possible, the sweat should be taken with the patient in the horizontal posture, as dizziness and faintness are less likely to occur. A drink of hot lemonade just before entering the bath often facilitates sweating. If the patient feels faint, or the pulse is disturbed, the sweat should be stopped. Discomfort of this sort is most apt to occur during the period of hyperemia of the skin, before perspiration has begun. The first few sweats are not as well borne as later ones, and it is advisable to use a lower degree of heat, and shorten the duration in the beginning. When possible, the patient should be kept at rest in bed during the two or three weeks he is taking his sweats. It has been my practice, when possible, to send the patient to the hospital during this period. When he has received daily sweats for ten days, the pressure is sufficiently lowered so that, during the remainder of the stay, sweats every second day are sufficient. During the intervals between these periodical sweats the patient may take one or two sweats per week at his home. Some patients, and this is especially true of those with extremely high blood-pressure, are very little affected by the sweat.

Attention should be called to the dangers of sweating. Some patients do not tolerate sweats. After a few days they complain of faintness, palpitation, and weakness. Under these circumstances the treatments should be given less frequently, the duration lessened, and, if these symptoms persist, entirely discontinued. At times the marked reduction of pressure following a sweat may cause alarming symptoms. After a sweat the patient should never be plunged in cold water. Such a precaution may seem unnecessary, yet it is a practice in some institutions after sweating a patient to turn on the cold spray.

In addition to the sweats, the skin should be kept active at all times.

Warm underclothing and careful protection from cold should be provided. In winter, when possible, these patients should seek a warm climate, as their dyspnea and other cardiac disturbances are more troublesome in cold weather. As a substitute for the sweat, the patient may go to springs where he can taken warm baths. These are beneficial, and in addition the patient is improved by the psychic influence of the cure.

Venesection.—This method is in common use for the reduction of high blood-pressure. The actual lowering of pressure following its use is slight, not greater than following a sweat. It is also transitory in character as within three or four hours the pressure returns to the previous level; nevertheless many patients report rather prolonged increased comfort after bleeding.[5] When taking blood-pressure before and after bleeding it is well to bear in mind that the excitement due to the prospective bleeding is sufficient to raise the pressure considerably above the normal, and when compared with the pressure after bleeding may give misleading information. In 5 patients with marked hypertension the blood-pressure was taken before and after bleeding, care being taken to avoid the psychic element. From 450 to 600 c.c. were withdrawn. The maximum reduction in pressure was 15 mm. One failed to show any lowering. Where there is threatened or fully developed uremia, or apoplexy, bleeding is indicated, provided the patient is not already anemic. Free catharsis and good elimination through the skin is an excellent substitute for venesection, and is less of a tax on the patient.

Vasodilators.—Vasodilators are purely palliative. Some observers have reported permanent lowering of the blood-pressure after their continued use. When this occurs, it is probably entirely independent of the drug, as remarkable daily variation in pressure may be noted in untreated cases. At times, for a period of several weeks and without apparent cause, the pressure may average much lower than the normal for that individual. Probably invariably when a patient has hypertension from chronic interstitial nephritis, it is permament. It may at most only be temporarily modified.

Dyspnea.—On account of the increased work thrown upon the heart by the high blood-pressure the patient should carefully avoid overexertion. He may be wisely advised to avoid exercise that makes him dyspneic, as rapid walking, stair climbing or hill climbing, walking against a strong wind, or taking much exercise in the open air in extremely cold weather. Systematic walking with frequent rests, on the other hand, may be beneficial by keeping the heart muscle in better condition. When, in spite of quiet on the part of the patient, dyspnea persists digitalis may be given. With cardiac incompensation pending, digitalis may be of value, notwithstanding the high pressure. The tincture may be used beginning with 10 minims (0.7 c.c.) three times daily, watching carefully for evidence of intoxication, as slowing or irregularity of the pulse. If no results are obtained, the dose should be increased to the point where a physiological effect is secured or evidence of intoxication develops. The development of marked cardiac dyspnea in a patient with hypertension is an ominous sign, and usually marks the beginning of the end. Rarely under these conditions can the compensation be completely restored. To relieve the heart of part of its burden, sodium nitrite, 2 gr. (0.12 gm.), three times daily, may be tried but rarely is beneficial. Free catharsis and sweats often give considerable relief. For the very trying nocturnal dyspnea, from which these patients suffer, next to rest opium is the most satisfactory remedy—the powdered extract in ½ to ¾ gr. (0.03 to 0.045 gm.) doses at bedtime—and usually finally the use of hypodermics of morphia. Under these conditions the ordinary hypnotics, as bromids, chloral, sulphonal, trional, and veronal, as a rule, by depressing the circulation, do more harm than good. The danger of morphinism must be considered, but, as severe nocturnal dyspnea is almost invariably the terminal stage of the trouble, the patient's comfort should be the first consideration. In the earlier stages the Nauheim treatment may be beneficial. Often associated with the dyspnea are attacks of acute pulmonary edema. Hypertension with failing cardiac strength is the most frequent cause of acute non-inflammatory pulmonary edema. Hypodermics of morphin are of value in cutting short the attack. Vasodilators are indicated, but rarely do any good. Atropin, on account of increasing the blood-pressure, may be harmful. In suitable subjects venesection is indicated, but rarely does any good. As attacks of acute pulmonary edema are often precipitated by the patient overtaxing in undressing or exposure to chilling, as a cold bath, cold room, or a cold bed, the proper measures should be taken to correct these conditions.

---

[5] The relief following bleeding, especially in patients with disagreeable head sensations, has never been satisfactorily explained. It is certainly not due to the decreased blood-pressure, as it may last for weeks and may occur where there is no reduction in pressure. Bleeding is not practiced freely enough in these patients.—Editor.

Headache.—Headache is very common in interstitial nephritis. While in some manner probably associated with the high blood-pressure, it is seldom relieved by the vasodilators. The coal-tar products most frequently give relief. Phenacetin, 7 gr. (0.5 gm.); antipyrin, 7 gr. (0.5 gm.), repeated if necessary in four hours, are reasonably safe.

Cerebral Hemorrhage.—Cerebral hemorrhage may be best treated by absolute rest, enforced if necessary by morphin. There is no value in special drug therapy. The skin should be properly cared for by proper cleansing and rubbing with alcohol. The bladder must be watched and catheterism resorted to if necessary. The bowels should not be disturbed for at least forty-eight hours after the hemorrhage, for fear of exciting further bleeding. Later it is important to obtain good elimination through the bowel and skin. After the lapse of two weeks massage and electricity tend to keep the muscles of the paralyzed extremities in better condition, and facilitate use of the part. When the patient recovers sufficiently to move the part he should be put through daily exercises in order to regain accuracy of control of his movements. For the arm movements touching the nose with the fingers, or touching a suspended object, piling up checkers, placing pegs in holes and numerous simple exercises of this character are of the greatest value in acquiring the proper control of the part. Massage is the most efficient measure to prevent contractures. A cerebral hemorrhage which partially disables the patient tends, perhaps unfortunately, to prolong his life. This enforced rest with freedom from worry and responsibility lowers blood-pressure and lessens the work of the heart, and the patient becomes more comfortable than he has been perhaps for years.

Summary.—The chief dangers in chronic interstitial nephritis are those incident to the high blood-pressure, for instance, cerebral hemorrhage and cardiac weakness. The amount of protein in the diet should not exceed the minimum body requirement, 60 to 70 gm. daily. It is probably a matter of little importance in what particular form the protein is taken, whether milk, meat, or eggs. The fats and carbohydrates may be taken freely, unless the patient tends to obesity. Marked restriction of fluids is inadvisable. A sufficient amount should be taken to make the daily output of urine between 1,500 and 2,000 c.c. (1½ to 2 quarts). Marked salt restriction is only necessary in the presence of edema. High blood-pressure is best treated by good elimination through the skin and bowel and by freedom from mental strain. When possible, a warm winter climate is advisable. When the pressure is extremely high or cardiac failure impending, the vasodilators may be tried. Of these the sodium nitrite in 2 gr. doses, three or four times a day, is most satisfactory. To protect the heart, the amount of physical exertion should be carefully regulated. Stair or hill climbing, fast walking, or running should be prohibited. When the cardiac compensation is broken, in addition to the complete rest, digitalis should be given in the form of a good tincture beginning with doses of 10 minims (0.7 c.c.) three times daily. For the pulmonary edema, use morphin sulphate hypodermically, ¼ gr. (0.015 gm.), bleeding, caffein citrate, oxygen inhalations.

MERCURIAL NEPHRITIS[6]

During the past four or five years there has been a very marked increase in the frequency of bichlorid poisoning. This has been largely due to the publicity given by the newspapers to cases of accidental poisoning. To those with suicidal intentions these articles gave the necessary information. The ease with which bichlorid could be obtained also furthered their object. Fifty per cent of the reported bichlorid poisonings, with suicidal intent, in the city of New York during the past twenty-two years have occurred during the past three years.

---

[6] For a more detailed discussion see the article on Bichlorid Poisoning, Volume III, page 634.

Usually within four days after the poison has been taken the patient develops marked symptoms of renal involvement. Lambert and Patterson, who have reported a series of cases, find that the mercury is regularly found in the stomach for several days after it was taken. This is not due to retention of the drug but to secretion into the stomach in the effort to eliminate it from the body. For this reason active treatment should be continued for several days or until chemical tests show the absence of bichlorid in the stomach contents, feces and urine. The following treatment has been outlined by Lambert and Patterson. In early cases the patients should be given the whites of several eggs. The stomach is then washed out thoroughly. The later treatment consists in washing out the stomach twice daily for one to three weeks or until tests show the absence of bichlorid. During the same period of time the colon is irrigated twice daily and the patient receives during the day 8 ounces of milk every two hours and every alternate hour 8 ounces of a mixture containing potassium citrate 1 dram; sugar 1 dram; lactose ½ ounce; lemon juice 1 ounce; water 16 ounces. Day and night during this period the patient is given by the drop method of rectal irrigation a solution containing 1 dram of potassium acetate to the pint of water, and it is important that this be continued day and night until on two successive days the urine has been free from mercury, or in case the test is not made, for a week in moderate cases and two weeks or more in severe poisoning. Summed up, this treatment consists in thorough and continued emptying of the intestinal tract, frequent washing of the stomach and very free fluid intake to facilitate flushing out the kidneys.

REFERENCES

NORMAL PHYSIOLOGY

Cushny, A. R. The Secretion of Urine, 197, London, Longmans, Green & Co.

Folin, O., and Berglund, H. Retention and Distribution of Amino-acids with Especial Reference to Urea Formation, Journ. Biol. Chem., li, 395, 1922.

Herringham, U. P. An Account of Some Experiments on the Toxicity of Normal Urine, Journ. Path. & Bacteriol., vi, 158, 1900.

Kossel, A., and Dakin, H. D. Weitere Untersuchungen über fermentative Harnstoffbildung, Ztschr. f. physiol. Chem., Strassb., xliii, 187, 1904.

Lewandowsky, M. Zur Frage der inneren Secretion von Nebenniere und Niere, Ztschr. f. klin. Med., xxxvii, 535, 1899.

Pearce, R. M. Influence of Kidney Extracts on Blood-Pressure, Journ. Exper. Med., xi, 430, 1909.

—— Depressor Substance of Dogs' Urine and Its Disappearance in Certain Forms of Nephritis, Journ. Exper. Med., xii, 128, 1910.

Tigerstedt, R., and Bergman, G. Niere und Krieslauf, Skand. Arch. f. Physiol., viii, 223, 1898.

Edema

Albu. Zur experimentel Erzeugung von Oedemen und Hydropsien, Virchow's Arch. f. path. Anat., clxvi, 87, 1901.

Bence, J. Experimentelle Beiträge zur Entstehung der nephritischen Oedem, Ztschr. f. klin. Med., lxvii, 69, 1909.

Bittorf. Zur Pathologie des Wasserund Salzstoffwechsels, Deutsches Arch. f. klin. Med., xciv, 84, 1908.

Blanch. Experimentelle Beiträge zur Pathogenese der Nierenwassersucht, Ztschr. f. klin. Med., lx, 472, 1906.

Blooker. Uber den Einfluss der Kochsalzzufuhr. auf die nephritische Oedem, Deutsches Arch. f. klin. Med., xcvi, 80, 1909.

Castaigne, J., and Rathelly, F. Etude experimentatal de l'action des Solutions de Chlorure de Sodium sur l'Epithélium Renal, Semaine méd., xxiii, 309, 1903.

Cohnheim, J., and Lichtheim, L. Uber Hydrämie und hydrämischen Oedem, Virchow's Arch. f. path. Anat., lxix, 106, 1877.

Dickson, E. C. Experimental Production of Chronic Nephritis in Animals, by the Use of Uranium Nitrate, Arch. Int. Med., iii, 375, 1909.

Fischer, M. Nephritis, John Wiley & Sons, 1912.

Georgopulos. Experimentelle Beiträge zur Frage der Nierenwassersucht, Ztschr. f. klin. Med., lx, 411, 1906.

Goldberg, J., and Hertz, R. Uber den Einfluss von Natriumbikarbonat und der Ausschiedung der Chlorid und des intravenos eingeführten Milchzuckers, Deutsches Arch. f. klin. Med., cxvi, 201, 1914.

Goodman, C. Use of Karell Cure in the Treatment of Cardiac and Renal Disease, Arch. Int. Med., xvii, 809, 1916.

Heincke, A., and Meyerstein, W. Experimentelle Untersuchungen über den Hydrop, bei Nierenkrankheiten, Deutsches Arch. f. klin. Med., xc, 101, 1907.

Magnus, R. Uber die Entstehung der Hautoedeme bei experimentelle hydrämeschen Plethora, Arch. f. exper. Path. u. Pharmakol., xliii, 250, 1899.

Meltzer, S. J. Edema, Am. Med., iii, 19, 151, 159, 191, 1904.

Pearce, R. M. The Production of Edema; An Experimental Study of the Relative Etiological Importance of Renal Injury, Vascular Injury, and Plethoric Hydremia, Arch. Int. Med., iii, 422, 1909.
Richter, P. F. Experimentelles über die Nierenwassersucht, Berl. klin. Wchnschr., xlii, 384, 1905.
Schlayer, Hedinger, and Takayasu. Uber nephritisches Oedeme, Deutsches Arch. f. klin. Med., xci, 59, 1907.
Timofeew, S. Zur Frage von der Pathogenese der nephritischen Oedeme, Arch. f. exper. Path. u. Pharmakol., lx, 265, 1909.
Widal. La Cure de Déchloruration, Bull, et mém. Soc. méd. d. hôp. de Paris, xx, 990, 1903.

EXPERIMENTAL NEPHRITIS

Christian, H. O. Experimental Nephritis, Boston Med. & Surg. Journ., clviii, 1908.
Christian, H. O., Frothingham, C., O'Hare, James. Studies in Nephritis, Am. Journ. Med. Sc., cl, 655, 1915.
Dickson, E. C. Experimental Chronic Nephritis in Animals by the Use of Uranium Nitrate, Arch. Int. Med., iii, 375, 1909.
Emerson, H. An Experimental and Critical Study of the Etiology of Chronic Nephritis, Arch. Int. Med., i, 485, 1908.
Lindeman. Uber das Wesen der toxischen Nephritis, Centralbl. f. allg. Path. u. path. Anat., xi, 308, 1900.
Lyon. Inflammatory Changes in the Kidney; an Experimental Study of the Action of Some Toxins and Poisons on the Kidney, and Also on the Spleen, Journ. Path. and Bacteriol., ix, 400, 1904.
Ophuls, W. Experimental Chronic Nephritis, Journ. Am. Med. Ass., xlviii, 483, 1907.
Pässler and Heincke. Versuche zur Pathologie des Morbus Brightii, Verhandl. d. Deutsch. path. Gesellsch., ix, 99, 1905.
Pearce, R. M. An Experimental Glomerular Lesion Caused by Snake Venom, Journ. Exper. Med., xi, 532, 1909.
Pearce, R. M., and Sawyer, H. P. Concerning the Presence of Nephrotoxic Substances in the Serum of Animals with Experimental Nephritis, Journ. Med. Res., xix, 269, 1908.
Schlayer and Hedinger. Experimentelle Studien über toxische Nephritis, Deutsches Arch. f. klin. Med., xc, 1, 1907.

HYPERTENSION

Alwens, W. Uber die mechanische Theorie der nephritischen Blutdrucksteigerung, Deutsches Arch. f. klin. Med., xcix, 137, 1909.
Arnold, H. D. High Frequency Electricity in the Treatment of Cardiac Disease, Boston Med. & Surg. Journ., clix, 233, 1908.

Austrian, C. R. The Viscosity of the Blood in Health and Disease, Johns Hopkins Hosp. Bull., xxii, 9, 1911.

Bachmann, E. Die klinische Verwertung der Viskositätsbestimmung, Deutsches Arch. f. klin. Med., xciv, 409, 1908.

Bütterman. Einige Beachtungen über Blutdruck bei Kranken, Deutsches Arch. f. klin. Med., lxxiv, 1, 1902.

Fellner, B. Klin. Erfahrungen über Vasotonin, Verhandl. d. Cong. f. innere Med., xxvii, 647, 1910.

Fraenkel, A. Adrenalingehalt des Blutes bei Chronischer Nephritis u. Morbus Basedowii, Arch. f. exper. Path. u. Pharmakol., lx, 395, 1909.

Janeway, T. C. Note on the Blood-Pressure Changes Following Reduction of the Renal Arterial Circulation, Proc. Soc. Exper. Biol. & Med., vi, 109, 1909. Ibid.

——— The Pathological Physiology of Chronic Arterial Hypertension and Its Treatment, Am. Journ. Med. Sc., cxxx, 50, 1907.

——— Management of Patients with Chronic Renal Disease, Ibid., cli, 157, 1916.

Kinnicutt, F. Nitroglycerin in the Treatment of Chronic Nephritis, N. Y. Med. Journ., xliii, 219, 1886.

Krehl, L. Pathologische Physiologie, 4th ed., 40, 1906.

Kretschmer, W. Uber die Etiologie der nephritischen Blutdrucksteigerung und vergleichend experimentelle Untersuchungen über blutzrucksteigernde Substancen, Verhandl. d. Cong. f. innere Med., xxvii, 731, 1910.

Loeb, A. Uber Blutdruck und Herzhypertrophie bei Nephritikern, Deutsches Arch. f. klin. Med., lxxxv, 348, 1905.

——— Klinische Untersuchungen über den Einfluss von Kreislaufsänderungen auf die Urinzusammensetzung, Deutsches Arch. f. klin. Med., lxxxiii, 452, 1905.

Loomis, H. P. The Limitations of the Value of Nitroglycerin as a Therapeutic Agent, Med. Rec., lxvii, 411, 1905.

Miller, J. L. Hypertension and the Value of the Various Methods for Its Reduction, Journ. Am. Med. Ass., liv, 1666, 1910.

Passler and Heineke. Versuche zur Pathologie des Morbus Brightii, Verhandl. d. deutsch. path. Gesellsch., ix, 99, 1905.

Schneider. Effect of Altitude on Blood Pressure, Am. Journ. Physiol., xl, 330, 1916.

Schur, H., and Wiessel, J. Uber die Adrenalin-Analoge, Wirkung des Blutserums von Nephritikern auf das Froschauge, Wien, klin. Wchnschr., xx, 699, 1907.

Stewart, D. Tolerance to Nitroglycerin, Journ. Am. Med. Ass., xliv, 1678, 1905.

UREMIA

Ascoli. Vorlesungen über Uraemie, Jena, 1903.

Ceconi, A. Kochsalz und Uraemie, München. med. Wchnschr., lvi, 497, 1909.

Feltz and Ritter. De l'Uremie Expérimentale, Paris, 1881.

Foster, N. B. Uremia, Arch. Int. Med., xv, 356, 1915.

Hartman, F. A. The Symptoms of Urinoid Poisoning, Arch. Int. Med., xvi, 98, 1915.

Leiter, L. Ibid., 1921.

Müller, F. Morbus Brightii, Verhandl. d. deutsch. path. Gesellsch., ix, 64, 1905.

Noorden, C. von. Handbuch der Pathologie des Stoffwechsels, l, 969, 1906.

Palmer, W. W., and Henderson, L. J. A Study of the Several Factors of Acid Excretions in Nephritis, Arch. Int. Med., xvi, 104, 1915.

Passler, H. Pathologie der Nierenkrankheiten, Verhandl. d. Cong. f. innere Med., xxiii, 325, 1906.

Peabody. The Acidosis of Chronic Nephritis, Arch. Int. Med., xvi, 955, 1915.

Pearce, R. M., and Sawyer, H. P. Concerning the Presence of Nephrotoxic Substances in the Serum of Animals with Experimental Nephritis, Journ. Med. Res., xix, 264, 1908.

Reiss, E. Zur. klinik und Einteilung der Urämie, Ztschr. f. klin. Med., lxxx, 97, 1914.

Soetbeer, F. Experimentelle Untersuchungen zur Urämie, Verhandl. d. Cong. f. innere Med., xxiii, 325, 1906.

Weiss, O. Uber die Wirkungen von Blut serum Injectionen im Blut, Arch. f. d. ges. Physiol., lxv, 215, 1896.

HEART HYPERTROPHY IN NEPHRITIS

Jores, C. Uber die Beziehung. der Schrumpnieren zur Herzhypertrophie vom pathologisch anatomischen Standpunkt, Deutsches Arch. f. klin. Med., xciv, 1, 1908.

Passler. Uber die Ursache und Bedeutung der Herzaffektion Nierenkranker, Samml. klin. Vortr., v, 408, 1906.

FUNCTIONAL KIDNEY TESTS

Albarran. Exploration des Functiones Rénales, 1905.

Blum, V. Die funktionelle Nierendiagnostik, Wien. klin. Wchnschr., xxiii, 685, 1910.

Cabot, R. C. The Diagnosis of Renal Functions, N. Y. Med. Journ., lxxxiii, 962, 1906.

Casper and Richter. Funktionelle Nierendiagnostik mit besonderer Berücksichtigung der Nierenchirurgie, Berlin, Wien, 1901.

Clairmont, P., and Herberer, H. Beiträge zur funktionellen Nierendiagnostik, Mitt. a. d. Grenzgeb. d. Med. u. Chir., xix, 316, 1909.

Emerson, C. P. Cylindruria, Journ. Am. Med. Ass., xlvi, 5, 1906.

Foster, N. B. Non-protein Nitrogen in the Blood, Arch. Int. Med., xv, 356, 1915.

Gertzen, M. Uber Zylindrurie und Nephritis, Deutsche med. Wchnschr., xxi, 1312, 1905.

Kapsammer. Nierendiagnostik und Nierenchirurgie, Wien, Leipz., 1907.

Klieneberger, C., and Oxenius, R. Uber Urine and Urinsedimente bei normalen Personen, bei rheumatischen Erkrankungen und nach der Einwirkung von Salizylpräparaten, Deutsches Arch. f. klin. Med., lxxx, 225, 1904.

McLean, F. C. Numerical Laws Governing the Rate of Excretion of Urea and Chlorid in Man, Journ. Exper. Med., xxii, 366, 1915.

Meltzer, S. J. Remarks on Albuminuria, Cylindruria, and Bright's Disease, N. Y. Med. Journ., lxxxiv, 171, 1906.

Mosenthal. Renal Function as Measured by the Elimination of Fluids, Salt and Nitrogen and the Specific Gravity of the Urine, Arch. Int. Med., xvi, 733, 1915.

Rowntree, L. G., and Geraghty, J. T. An Experimental and Clinical Study of the Functional Activity of the Kidneys by Means of Phenolsulphonephthalein, Journ. Pharmacol. & Exper. Therap., i, 579, 1910.

Schlayer and Takayasu. Untersuchungen über die Funktion kranker Nieren beim Menschen, Deutsches Arch. f. klin. Med., ci, 333, 1911.

Schreiber, J. Uber Abdominell-palpatorische Albuminurie, Deutsches Arch. f. klin. Med., xcvii, 1, 1909.

Tileston, W., and Comfort, C. W. The Total Non-protein Nitrogen and the Urea of the Blood in Health and Disease, etc., Arch. Int. Med., xiv, 621, 1914.

Diet in Nephritis

Arnold, H. D. The Importance of a Low Protein Diet in Nephritis, Journ. Am. Med. Ass., lv, 2193, 1910.

Barger and Walpole. Isolation of Pressor Substance in Putrid Meat, Journ. Physiol., London, xxxviii, 343, 1910.

Blatherwick, N. R. The Specific Rôle of Foods in Relation to the Composition of the Urine, Arch. Int. Med., xiv, 409, 1914.

Dale and Dixon. The Action of Pressor Aminos Produced by Putrefaction, Journ. Physiol., London, xxxix, 25, 1910.

Epstein. Medical Clin. North America, 1921.

Frothingham, C., and Smullen, W. G. A Study of Different Nitrogenous Diets in Nephritis, Arch. Int. Med., xvi, 204, 1915.

Hesse, E. Der Einfluss des Rauchens auf den Kreislauf, Deutsches Arch. f. klin. Med., lxxxix, 568, 1906.

Kaufman and Mohr. Beiträge zur Diätetik der Nierenkrankheiten, Ztschr. f. klin. Med., xliv, 441, 1902.

Kovesi, G., and Roth, Schulz, W. Therapie der Nierenentzündungen, Berl. klin. Wchnschr., xli, 632, 1904.

Leube, W. Handbuch der Therapie Inneren Krankheiten, Penzoldt and Stintzing, vii, 218, 1898.

Lusk, G. The Science of Nutrition, 2d ed., 1910.

Mann, J. D. Pharmacology of Tobacco Smoke, Brit. Med. Journ., ii, 1673, 1908.

Noorden, C. von. Diseases of Metabolism and Nutrition, Nephritis, ii, 1903.

Smillie, W. G. Potassium Poisoning in Nephritis, Arch. Int. Med., xvi, 330, 1915.

Strauss, H. Diätbehandlung innerer Krankheiten, Berlin, 1908.

White, H. On the Influence of Various Diets Upon the Composition of the Urine and the General Condition of Patients Suffering from Chronic Bright's Disease, Med.-Chir. Tr., lxxvi, 279, 1894.

Diuretics

Christian, H. C. The Effect of Theobromin Salicylate in Acute Experimental Nephritis, Arch. Int. Med., xiv, 327, 1914.

Cushny, A. R. Pharmacology and Therapeutics, 5th ed., 1910.

Eichmüller. Herz-und Gefaess-wirkung des Digalen bei gesunden und kranken Menschen, Berl. klin. Wchnschr., xlvi, 1677, 1909.

Hedinger, M. Uber die Wirkungsweise von Nieren und Hermitteles auf kranke Nieren, Deutsches Arch. f. klin. Med., c, 305, 1910.

MacNeder, W. D. A Study of the Action of Various Diuretics in Uranium Nephritis, Journ. Pharmacol. & Exper. Therap., iii, 423, 1912.

Meyer, H. H., and Gottlieb, R. Die experimentelle Pharmakologie als Grundlage der Arzneibehandlung, 2d ed., 1911.

Schlayer. Untersuchungen über die Funktion kranker menschlicher Nieren, Verhandl. d. Cong. f. innere Med., xxvii, 794, 1910.

Prophylaxis in Nephritis

Emerson, H. An Experimental and Critical Study of the Etiology of Chronic Nephritis, Arch. Int. Med., i, 485, 1908.

Friedenwald, J. The Physiological Effects of Alcohol on Rabbits, Journ. Am. Med. Ass., xlv, 780, 1905.

Klieneberger, C. Quecksilberschmierkuren und ihre Einwirkung auf die Harnorgane, Ztschr. f. klin. Med., lviii, 481, 1906.

Klieneberger, C., and Oxenius, R. Uber Urine und Urinsedimente bei normalen Personen, bei rheumatischen Erkrankungen und nach der Einwirkung von Salizylpräparaten, Deutsches Arch. f. klin. Med., lxxx, 225, 1904.

Mühlig, F. Ein Fall von Nephritis parenchymatösa syphilitica, München. med. Wchnschr., l, 505, 1903.

Siegel, W. Experimentelles zur Frage der Erkältungsnephritis, Verhandl. d. Cong. f. innere Med., xxv, 510, 1908.

Wagner, M. Uber parenchymatöse Nephritis bei Lues, München. med. Wchnschr., xlix, 2073, 1902.

Climate in Nephritis

Loew, O. Akklimatisierung, München. med. Wchnschr., lv, 1585, 1908.

Sewall, H. Recent Additions to Our Knowledge of the Physiological Influence of Lowered Barometric Pressure, Internat. Clin., iv, 16.

Staubli, C. Beiträge zur Kenntnis des Einflusses des Hochgebirgsklimas, Verhandl. d. Cong. f. innere Med., xxvii, 695, 1910.

Strauss, H. Klima und Nierenkrankheiten, Deutsche med. Wchnschr., xxiv, 2059, 1908.

Rest

Dunlap and Paton. On the Influence of Muscular Exercise on Metabolism, Journ. Physiol., London, xxii, 68, 1897.

Krummacher. Einfluss der Muskelarbeit auf die Einweisszersetzung, Arch. f. d. ges. Physiol. (Pflüger's Arch.), xlvii, 454, 1890.

Effect of Fluids on Nephritis

Kovesi and Roth-Schulz. Uber Störungen der Wassersezermerenden Tätigkeit diffus erkrankter Nieren, Berl. klin. Wchnschr., xl, 32, 1900.

Lindermann. Die Konzentration des Harns und Blutes bei Nierenkrank. Deutsches Arch. f. klin. Med., lxv, i, 1899.

Miller, J. L., and Williams, J. L. Am. Journ. Med. Sc., clxi, 327, 1921.

Mohr, S., and Dapper, C. Beiträge zur Diätetik der Nierenkrankheiten, Ztschr. f. klin. Med., l, 377, 1903.

Nagelschmidt, F. Uber Alimentäre Beeinflüssung des osmotischen Druckes, Ztschr. f. klin. Med., xlii, 274, 1901.

Sodium Chlorid Restriction

Blooker, J. W. Uber den Einfluss der Kochsalz Zufuhr auf die nephritischen Oedeme, Deutsches Arch. f. klin. Med., xcvi, 80, 1909.

Kovesi, G., and Roth, Schulz, W. Therapie der Nierenentzündungen, Berl. klin. Wchnschr., xli, 632, 1904.

Magnus-Levy, A. Dehydration by Dietetic Measures, Journ. Am. Med. Ass., lv, 2139, 1910.

Miller, J. L. The Elimination of Chlorids in Nephritis, Tr. Ass. Am. Physicians, 1905.

Noorden, C. von. Handbuch der Pathologie des Stoffwechsels, 2d ed., i, 1000, 1906.

Vaughan, V. C. Value and Limitations of Salt-free Diet and Restriction of Fluids in Nephritis, Journ. Am. Med. Ass., liii, 1289, 1909.

Widal, F. Javal. La Chloruréurie et la Cure de Déchloruration, Journ. de Phys., v, 1107, 1123, 1903.

Sweats in Nephritis

Austin and Miller. Influence of Sweat Baths on Non-protein Nitrogen in the Blood, Journ. Am. Med. Ass., lxxx, 944, 1914.

Bendix, E. Uber Wechselbeziehungen zwischen Haut und Nierentätigkeit., Deutsche med. Wchnschr., xxx, 233, 1904.

Brieger, L., and Diesselhorst, G. Untersuchungen über den menschlichen Schweiss, Deutsche med. Wchnschr., xxix, 167, 421, 1903.

Kovesi, G., and Roth, Schulz W. Therapie der Nierenentzündungen, Berl. klin. Wchnschr., xli, 632, 1904.

Plaggemeyer, H. W., and Marshall, E. K. A Comparison of the Excretory Power of the Skin with That of the Kidney through a Study of Human Sweat, Arch. Int. Med., xiii, 159, 1914.

Strauss, H. Uber Nierenentlastung durch Schwitzen, Deutsche med. Wchnschr., xxx, 1236, 1904.

Uremic Dyspnea and Acute Pulmonary Edema

Miller, J. L., and Matthews, S. A. A Study of the Mechanical Factors in Experimental Acute Pulmonary Edema, Arch. Int. Med., iv, 356, 1909.

Riesman, D. Acute Pulmonary Edema, with Special Reference to a Recurrent Form, Tr. Ass. Am. Physicians, xxi, 155, 1906.

Sahli, H. Zur Pathologie und Therapie des Lungensödems, Arch. f. exper. Path. u. Pharmakol., xix, 431, 1885.

Welch, W. H. Zur Pathologie des Lungenödems, Virchow's Arch. f. path. Anat., lxxii, 375, 1878.

Mercuric Chlorid Poisoning

Foster, N. B. Mercury Nephritis, Arch. Int. Med., xv, 754, 1915.

Lambert, S. Poisoning of Mercuric Chlorid and Its Treatment, Arch. Int. Med., xvi, 805, 1915.

Vogel, K. M., and Lee, O. L. Detection of Mercury in the Excretions, Journ. Am. Med. Ass., lxii, 532, 1914.

CHAPTER XXV

AMYLOID DISEASE OF THE KIDNEY

JOSEPH L. MILLER

The treatment of amyloid disease of the kidney is purely prophylactic in the treatment of diseased conditions responsible for its development. The two most important causes are chronic suppurative processes and tertiary syphilis. Tuberculosis, with secondary pyogenic infection, is a common cause. Rarely is tuberculous infection alone responsible for its development. Chronic suppurative bones and joints, liver abscess, and empyema are common etiological factors. Bull was able to demonstrate amyloid changes in the kidney three weeks after the development of an acute liver abscess. A number of other etiological factors have been held responsible for the development of amyloid diseases. They occur so infrequently and their actual relation to the process is so doubtful that they need not be considered.

The best preventive measures are the prompt and thorough drainage of pus collections and the proper treatment of syphilis. There is no reason to believe that the use of nitric acid, as recommended by Budd, has any specific action. As in many of those cases there are associated amyloid changes in the intestine, with consequent diarrhea, treatment by diet and the remedies used in diarrhea are indicated.

There may be extensive amyloid changes in the kidney with negative urinary findings, and frequently the albumin and casts are due to the development of a parenchymatous nephritis in addition to the amyloid. Uremia probably never develops from a pure amyloid kidney. Hirose in a series of 59 cases found that the presence of amyloid in the kidney was always associated with evident chronic nephritis. In only one of 15 cases where the blood-pressure was taken was it above normal. Edema, when present, is the result of other causes—weak heart, anemia, cachexia, or an associated nephritis. There is never a high blood-pressure resulting from pure amyloid degeneration of the kidney. Hypertension is suggestive of an interstitial nephritis in addition to the amyloid. As there are so few renal symptoms, the treatment should be directed at the primary disturbance. The anemia should be treated by diet and iron. When there is evidence of cardiac weakness rest and digitalis are indicated.

Every patient with a chronic disease becomes more or less neurasthenic. He develops a group of symptoms purely nervous in character. In fact, in many mild cases of chronic nephritis, most, or all, of the subjective disturbances may be of this character. It is well to recognize the real nature of these symptoms, and to relieve the patient by the assurance that they are not due to his kidney condition. The importance of psychotherapy is well illustrated in those patients who take up Christian Science. The subjective improvement is very marked; the patient is encouraged and his neurotic symptoms vanish, although the condition of the kidney is unchanged.[1]

The Kidney of Pregnancy.—With the more recent investigation of the toxemia of pregnancy, it has been quite definitely determined that the kidney is less responsible than was formerly claimed. The appearance of eclampsia in cases of vesicular mole would indicate that the fetus is not essential to its development. Efforts to obtain a toxic substance from the placenta have given negative results. The toxic substance, presumably a ferment, has an autolytic action on the tissues of the body, more especially the liver. In this organ there is early capillary thrombosis, especially marked in the periphery of the lobule, with destruction of the liver cells and marked tendency to hemorrhage. All the other organs may be involved, usually to a less degree. We are dealing here with a generalized intoxication. When the kidney is markedly affected by this toxin, albumin and casts appear in the urine. It is not a localized process in the kidney like that present in acute nephritis. The eclamptic seizure, while resembling uremia, is probably of entirely different origin and not due to disturbed kidney function. Many cases of eclampsia fail to show evidence either clinically or at autopsy of renal involvement. In some toxemias of pregnancy there is actually an acute nephritis. In eclampsia, however, nephritis is rare but hypertension the rule. The early recognition of the toxemia is very important. When advanced to the point where convulsions develop the results of treatment are much less favorable. Headache, restlessness, disturbance of vision, high blood-pressure, or albumin and casts in the urine may be taken as evidence of intoxication. The protein food should be reduced to a minimum, and increase the elimination through the kidney, skin, and bowel. The diet should consist of milk not to exceed a quart, and cereals, cooked fruits and other carbohydrate foods. If there is no edema the kidney should be thoroughly flushed by free water drinking. When hypertension is present, venesection with withdrawal of a pint of blood may be of some value. Free sweating is indicated, and until the symptoms are alleviated the patient may take two good sweats daily. The bowel elimination should be increased by the use of saline cathartics. When, in spite of these measures, the intoxication increases, the uterus should be emptied. Stroganoff has employed the following method of treatment of eclampsia with good results: the patient is isolated in a quiet, darkened room. The convulsions are controlled by the use of morphin hypodermically every two or three hours. In addition chloral hydrate, 10 gr. (0.65 gm.) in milk, every hour until five or six doses are given, and if convulsions still tend to appear they must be controlled by chloroform. The patient inhales oxygen freely. Elimination is increased by free water drinking and sweating. As a rule, within twenty-four hours after such treatment is begun labor sets in. When the symptoms do not abate within twenty-four hours, labor must be induced, or the uterus emptied by cesarean section. Stroganoff lays much stress on keeping the patient quiet, and as free from pain as possible.

---

[1] See article on the Toxemias of Pregnancy, Volume III, Chapter 46.

As a rule, after delivery the hypertension subsides and the individual apparently returned to normal health. Not infrequently, however, it persists. The author has had under observation for several years a number of patients where the hypertension persisted, all without evidence of kidney involvement or retention of urea.

REFERENCE

Hirose. Johns Hopkins Hosp. Bull., xxix, 191, 1918.

CHAPTER XXVI

PYELITIS

FRANK SHERMAN MEARA AND ALFRED TOWNSEND OSGOOD

Since pyelitis, pyonephritis, pyelonephritis, abscess of the kidney, perinephritis are merely terms, designating the particular locality in which identical processes from identical causes are operative, all of them suppurative inflammations of one or the other or a combination of structures of the kidney, no effort to consider them individually will be made.

The one absolute necessity for the production of a suppurative inflammation of the kidneys is the presence of pyogenic organisms. Unless the numbers are very great or the virulency of the organisms intense, other contributory factors are demanded to occasion the attack.

The organisms most responsible are the Bacillus coli communis, the Streptococcus pyogenes, and the Staphylococcus pyogenes aureus, yet the Staphylococcus pyogenes albus, Streptococcus mucosus capsulatus, pneumococcus, gonococcus, Bacillus typhosus or paratyphoid bacillus, may be the cause.

The importance of bacillus coli in these infections is constantly more keenly appreciated, and this applies in particular to young children and infants. Colon cystitis, pyelitis, pyelonephritis, and general infection in early life form a new chapter in medicine. Females are much more commonly affected than males.

The routes of invasion of pathogenic organisms are four: (1) by the blood-stream, sometimes called descending infection, the route by which most primary infections occur, and referred to as hematogenous; (2) through the urinary tract, ascending infection, the common route of secondary infections, and sometimes referred to as urogenous; (3) from bladder to kidney by way of the lymphatics of the ureter, of the pelvis and of the kidney substance, or (4) by direct transmission from the colon to the kidney, by the lymphatics.

Unless the virulence and numbers of the pathogenic organisms are great, one must assume, and, indeed, careful search is increasingly rewarded with the finding of, local disturbance in the kidney that renders it a locus resistentiæ minoris. In hematogenous infection no doubt the damage done the kidney by the secretion of the toxins of disease has lowered resistance locally and the onset of pyelonephritis or suppurative processes in the parenchyma of the kidney in convalescence so commonly observed favors this view.

It is important from a therapeutic point of view to consider the rôle of constipation and colon inflammation in these infections, because they favor a passage of the colon bacilli into the blood and lymphatics, enhance the virulency of the germ, and, by compression of the ureter, especially the right, by the distended gut, bring about a degree of urinary stasis which contributes powerfully to infection.

The hematogenous infections are commonly bilateral, but by no means always so, for a lesion of some kind existing in only one kidney may determine infection there while the sound kidney escapes.

In ascending or urogenous infection perhaps the most important factors are those that contribute to urinary stasis and abnormal patency of the ureteral orifice, to permit a back flow from the bladder. Bladder spasm may do this and even force the ureteral orifice.

This interference to the urinary flow in any part of its course, local lesions, and pathogenic microörganisms are the three factors the therapeutist has to keep in mind, with a view to prevention or removal. Renal calculus, inducing damage to kidney substance, pelvis, ureter, or bladder, and easily obstructing the passages, is always to be early considered; hydronephrosis, tuberculous kidney, new growths, stricture, inflammation or blockage by calculus or tumor, or growth in pelvis or ureter along its course or at its orifice, constipation as just mentioned, chronic cystitis, "acute cystitis with severe tenesmus and violent expulsive efforts" (Brewer), prostatic enlargement, strictures of the urethra are also to be considered. Colon bacillus infection occurs frequently during pregnancy, and Wildbolz notes this infection in newly married women and has termed it "defloration pyelitis." The condition is not uncommon after operative procedure, especially about the appendix.

The symptomatology of pyelonephritis is often so dominated by the condition to which it is secondary, as, for instance, by typhoid, grip, or pneumonia in the descending infection or by prostatitis, cystitis or calculus in the ascending infection, that the involvement of the kidney may not be suspected unless the examination of the urine has elicited attention; or, again, complications or terminal events, such as uremia or peritonitis from rupture, may be attributed to contracted kidney or appendicitis if the urinary examinations be neglected.

The symptoms referable to the pyelonephritis itself may be divided into those of local and general significance. The general symptoms are those of fever and toxemia; the local those of pain and urinary disturbance. Tenderness in the costovertebral angle is the most constant and characteristic sign. The pain is often absent or slight, a soreness or stiffness in the lumbar region, sometimes more marked and interpreted as lumbago. At times the pain is felt in the abdomen, radiating along the ureter and into the thigh, genitals, and perineum, like the pain of renal calculus, or, if on the right side, may be thought that of appendicitis, but it should be emphasized that in many cases there is no tenderness, no pain, no urinary disturbance subjectively; general symptoms, fever and toxemia and purulent urine are the only symptoms. These patients are often gravely ill and the absence of local signs referable to a kidney misleads in diagnosis. In such cases, no indication as to whether one or both kidneys are involved may be gained except through the aid of special examination with the cystoscope and catheterism of the ureters. This is now commonly carried out when necessary, even in very young children, females especially. It may be stated that no diagnosis of pyelitis is definite until it has been demonstrated that pus is produced in the kidney pelvis. This demonstration is only made by the catheterism of that renal pelvis. In all cases of this nature a very careful search for the bacillus tuberculosis should be made and other bacteria should be identified by stain and culture, for it is the type of bacterium which indicates the plan of treatment to be pursued. The question always present is whether renal or ureteral calculus complicates the situation or has given rise to the inflammation in the kidney. X-ray examination is therefore imperative.

The urinary disturbance may be expressed by polyuria, frequency of micturition, or slight pain after voiding, while the urine may attract attention by its cloudy aspect, or much more rarely by the red tinge of a slight hematuria. The examination determines the presence of pus and bacteria. Not only should the urine be examined in the routine manner, but the functional integrity of the kidney should be determined by tests now universally employed; namely, concentration tests, the phthalein test and estimation of retention of urea and other metabolites in the blood.

TREATMENT

The management and care of a patient suffering from pyelitis or pyelonephritis are modified by the acuteness or severity of the process. If the attack is acute or the patient suffers from an exacerbation of the process in a chronic infection, or, in other words, if there is a constitutional reaction to the local lesion, evidence of toxemia, the patient should be treated like all cases of acute infection by being put to bed at rest with the best nursing obtainable. The room chosen should be large, well lighted, well ventilated, free from encumbrances to gather and distribute dust, near a bathroom, and, if possible, leading from a porch or veranda. The bedstead should be of iron or brass, which can be readily kept clean, half or three-quarters width to facilitate handling, with woven wire springs and a firm mattress. The bed should be neatly made, with the minimum of clothes for the comfort of the patient. Rest is one of the most important factors in the treatment, and too often taken as a mere phrase. It economizes the patient's strength, facilitates repair of tissues, lessens the energy demands to be supplied by food, and should demand the same careful attention to detail in its attainment that is given to diet and drugs. Rest requires a well-made bed, a proper toilet, a quiet environment, a rigid exclusion of all causes for worry, anxiety, and useless conversation.[1]

A daily sponge bath for cleanliness, more frequent sponges for fever, care of the mouth, attention to the bowels, fresh air in the room, or removal of the bed to the outside air of the veranda as much as possible in the twenty-four hours are all-important items in the treatment. When patients are free from fever they are better out of bed, the amount of exercise and work allowed them to depend on their reaction to the same in terms of well-being and weight. I am firmly convinced that for these patients nothing is of greater value than fresh air, and I warmly advocate the practice of sleeping in the open air at all times of the year, even in the northern climates; of course, with due precaution to the means of protection while in the open.

These patients may take warm baths for purposes of cleanliness and a short, sharp shower bath afterward if the reaction is prompt and satisfactory. Sea bathing introduces the risk of too long exposure, chilling of the damp clothes on coming out, and possibly of undue fatigue, indeed, offers too many risks of lighting up a smoldering process.

Diet.—In acute cases the diet should be bland and non-irritating, but should be sufficient. Certain fundamental principles must be kept in mind in this condition, as in all acute infectious diseases, namely, that the food demands of a febrile patient at rest are not only as great as that of a well man at rest but more in many instances, for pyrexia above 102° F. will ordinarily increase the caloric output some 25 per cent, while the so-called toxic effect on tissue induces even greater katabolic processes. One may take 40 calories per kilo of body weight as a fair working estimate. This would require about 2,800 calories for a man of average weight of 70 kilos, or 154 pounds; or, more roughly, the figure 3,000 may be aimed at for the average man. This food should contain the lower limit of protein need, which may be taken at 70 to 80 grams, and the protein, it must be remembered, is the only food with which the kidney is concerned as an eliminative organ, if we exclude water of combustion and salts.

Milk has been shown by practical experience, and approved by theoretical considerations, as the best food for the basis of the dietary. If milk, however, is used exclusively, one of two things happens: too much protein is ingested to obtain enough calories to meet energy requirements; or, if the protein is kept within proper limits, the patient is underfed; for 1 quart of milk contains 640 calories (700 calories to the liter) and 35 gm. of protein. Between 4 and 5 quarts would be required for the 3,000 calories and that would furnish between 140 and 175 grams of protein. To obviate this difficulty 2 quarts of milk, furnishing 1,280 calories and 70 gm. of protein, may be used, fortified with milk sugar; while toast, cereals with sugar and cream, make up the balance. An ordinary slice of bread (1½ oz.) furnishes 100 calories; a pat of butter (0.4 oz.) 100; a dish of cereal 100; 2 ounces of cream 100; every ounce of sugar 120. If an ounce of milk sugar were added to each glass of milk in the 2 quarts nearly 1,000 calories would thus be added. Other forms of milk, such as buttermilk, or acidified milks, in which the fat is richer than in ordinary buttermilk, may be used, but the caloric value must be appreciated as being lower than the straight milk. For drinks, water, cereal waters, lemonade, and weak tea or coffee may be given, and sugar added to any of the drinks, of course, has a dietetic significance.

---

[1] I have a distinct impression that some of these patients do better in a Gatch bed in a semisitting posture.—Editor.

In the chronic conditions a sufficiency of diet is, again, all-important. Fats and carbohydrates should take a liberal part in it. It should be ordered along much the same line as in chronic nephritis. It includes meats in moderation, fish, eggs, vegetables, cereals, breadstuffs, simple desserts. It should have variety to preserve appetite. The articles to be excluded are all substances which irritate the kidney. They include all condiments, pepper, mustard, etc., radishes, parsley, onions, salted, dried, smoked, potted, deviled, or otherwise preserved meats, rich gravies, and pastries. Alcohol should be rigidly excluded. As important as in nephritis is the insistency on simplicity. Three meals a day, five hours between, only one hearty meal, only two or three courses, and one or two vegetables. All food fresh, simply prepared, and well cooked.

Water should be drunk freely, unless the condition of the circulation or kidney parenchyma forbids. When in an acute or chronic process the parenchyma of both kidneys is involved, their functional integrity should be determined by such tests as the phenolsulphonephthalein of Rowntree and Geraghty and the determination of retained metabolites in the blood, such as urea-nitrogen and creatinin-nitrogen, and the diet regulated accordingly, as in acute nephritis. If the parenchyma of one kidney alone is involved as determined by elimination of phthalein and metabolites, on ureteral catheterism, it is still desirable to economize in protein and salt in the diet.

Local Treatment.—Measures of physical therapy are directed to the kidney region with a view to relieving pain and discomfort and to a possible influence upon the inflammatory process.

Pain is rarely as intense as in renal colic, though the pyelonephritis may be the result of the nephrolithiasis, and is often interpreted as a slight lumbago, or the patient may complain of mere stiffness. For the relief of pain we have recourse to hot fomentations, poultices, hot-water bag, or hot bath of short duration and as hot as comfortably borne; in some cases, cold by applying the ice-bag (for the technic of these procedures see Nephrolithiasis). Dry cups may be applied, though, if there were a great deal of paranephritis, wet cups might afford relief. One may use small medicine glasses, egg cups, wine glasses. The skin over the kidney region should be cleansed, and as many cups as the part will accommodate applied.

In some cases the pain may be severe enough to demand the administration of a drug, and this should be small amounts of coal-tar, such as phenacetin, gr. v (gm. 0.3), or acetanilid, gr. iii (gm. 0.2), every half hour or hour for three doses; or, if this fails, morphin as a suppository, gr. 1/4 to gr. 1/3 (gm. 0.015 to 0.020), hypodermically, in doses of gr. 1/8 to gr. 1/4 (gm. 0.008 to 0.015), the method and dose depending on the severity of the pain.

Bowels.—The condition of the bowels should be a matter of concern, for neglect may entail not only the usual results of malaise, headache, impaired appetite, load the products of fermentation and putrefaction absorbed from the bowel upon the blood, but constipation and its results enhance the virulency of the Bacillus coli, so often the microörganism concerned, facilitate its passage into the lymph or blood vessels, while the distended gut by pressure on the ureter encourages stasis of its contents and forwards the process of infection. In the chronic cases the diet should contain enough coarse material, cellulose or seeds of fruits, to relieve constipation, or mild cathartics, such as compound licorice powder or saline waters, may be used. In the more acute cases, salts or enemata, or better yet irrigation of the bowel, twice a day at first, with plain hot water (120° F. or more, as borne) three gallons, may be employed. Use a catheter, male, about 20 F. The reservoir may be a fountain syringe or other container held three feet or so above the level of the patient.

Diuretics and Urinary Antiseptics.—Whenever a deleterious substance enters the body or appears in the body two mechanisms for protection are immediately set at work: first, one to render innocuous or destroy the deleterious substance, either by chemical combination or the elaboration of immune bodies; and, second, by elimination. The three great eliminating organs or emunctories are, then, or ought to be, objects of special solicitude in any infection, and their functions jealously preserved or encouraged. We have emphasized the care of the skin and bowel, but in the disease under consideration the third great emunctory is itself the site of morbid processes, yet must perform its share in the work of elimination. The importance of eliminating from the diet all irritating substances that might further burden the kidney has been touched upon, but it is equally important to remember to exercise the same precaution in the choice of drugs and exclude any that might cause the inflammation to flare up, remembering that even drugs applied to the skin may affect the kidney, as in the application of cantharides.

The best diuretic is water, and its ingestion should be encouraged in the form of plain water or water flavored with fruit juices or a mild diuretic like Imperial Drink (cream of tartar, ℥ss to Oiii boiling water, with sugar and lemon peel to the taste, to be taken ad libitum); or a physiological salt solution (roughly, a teaspoonful of salt to a pint of water) may be given by the bowel, as an enteroclysis (see Nephrolithiasis), a slow irrigation, or a Murphy drip, using the term as a slow continuous irrigation.

My own apparatus consists of a thermos bottle, with a perforation in the top, through which a tube connects with the irrigation tube, while a perforation in the bottom of the inverted bottle admits the air. This apparatus keeps the temperature even with the least trouble. The tube from this bottle is pinched by a cock in such a way as to regulate the flow from the bottle. One may use any kind of a container. The rectal tube may be hard rubber, glass, or soft rubber, but, in case of the former, the shape should be such as to avoid continuous pressure against the wall of the rectum. The temperature of the physiological salt solution used is 100° F. The time varies from two to three hours to twenty-four hours in the day, depending on results and the manner in which the patient bears it. In severe toxemias, or in threatened anuresis, a hypodermoclysis or an infusion may be indicated. The effect of the diuresis is a flushing out of the pelvis of the kidney, ureters, and bladder, a kind of natural irrigation.

The urine may be acid or alkaline. In the case of colon bacillus or typhoid pyelitis, the urine is usually acid; in the streptococcic and other forms of infection, which are secondary to an infection lower down, it is usually alkaline. Changing the reaction of the urine seems to afford a less suitable medium to the bacterium concerned, so, in case of an acid urine, alkaline salts, such as bicarbonate of soda, citrate of potash, or acetate of potash, or acid potassium tartrate, that is, cream of tartar, may be used in doses of gr. xv to gr. xxx (gm. 1 to 2), singly or in combination, every two hours until the urine is alkaline, and then the dose reduced in amount or frequency until only enough is given to keep the urine alkaline. If the urine is alkaline, benzoic acid, preferably as one of its salts, sodium or ammonium, or salicylates of sodium or aspirin, acetylsalicylic acid, in doses of gr. x to gr. xx (gm. 0.6 to 1.3) or more every four hours, or acid sodium phosphate gr. x (0.66 gm.) every four hours, often with urotropin gr. x q.4.h., is indicated until the object is accomplished and then only enough to keep the urine alkaline.

Benzoic acid, eliminated as hippuric acid, and salicylic acid, eliminated as salicyluric acid, are antiseptic in their qualities, but the one drug which at present seems the most efficacious as an antiseptic is hexamethylenamin, chemically, hexamethylenetetramin, which, in its own form or that of its derivatives, is known by several trade names, urotropin, helmitol, formin, etc., a regrettable multiplicity of nomenclature, that has led the careless on occasion to change from one to the other on the supposition of a difference in their chemical constitution and pharmacology.

The activity of the drug seems to be dependent on the liberation of formaldehyd, which is said to occur in the course of its elimination by the urinary tract. It is maintained that this liberation of formaldehyd occurs only in an acid medium, and, guided by this belief, some clinicians insist on the simultaneous administration of benzoic acid or its salts or acid sodium phosphate. It is worthy of trial before urotropin medication is abandoned as useless in a given case. The dose of urotropin should be gr. xxx to gr. lx (2 gm. to 4 gm.) a day, gr. v to gr. x (0.33 to 0.66 gm.) in water four times a day or every four hours.

The drug may be administered for a long time without evidence of harm, though occasionally in large doses, or in patients peculiarly susceptible, hematuria may occur or gastric unease, bladder distress, and abdominal pain, and diarrhea. These symptoms subside promptly on withdrawal of the drug. All bacteria are not equally susceptible to its influence; the typhoid bacillus and the bacillus coli seem the most so. My impression is that in the severe forms of bacillus coli infection in adults, I have had better success with alkalis than with urotropin.

Local Treatment.—This must be carried to the renal pelvis by the ureteral catheter. Many cases are relieved by the simple insertion of the ureteral catheter up to the renal pelvis. This affords drainage and supplants the physiologic pelvic and ureteral peristalsis. Irrigation of the renal pelvis through this catheter may be done with salt solution or with any mild antiseptic solution. This procedure must be carried out only by an experienced specialist to whom the details of slow injection of proper quantities and all the technic are well known.

Vaccine Therapy.—Studies in immunology have brought to the practitioner hope in abundance and results in some measure.

In pyelonephritis there are two conditions which may prevail that would invite to vaccination: first, a persistency of the local process, a pyelitis or pyelonephritis that has resisted the older measures and is becoming chronic; and, second, the septicemia that may result. The former alone offers much promise to the effort; in the latter condition it is a *dernier ressort.* Numerous reports have been made on vaccine therapy, especially in the colon bacillus cases, with no unanimity of opinion as to its value; but, on the whole, I think the statistics are encouraging.

A case of my own, due to Streptococcus mucosus capsulatus, mixed with pyocyaneus, in whom a nephrectomy was done three years before his death, owed, in my opinion, a freedom of constitutional symptoms, a gain in weight to the maximum of his life, and strength to conduct a large business with heavy responsibilities during nearly all the interval, to the use of autogenous vaccines, but in his case, as has been repeatedly observed, the urine made but little improvement, in spite of the great improvement in well-being. In all cases, however, as in this, open-air sleeping, careful hygiene, and diet must be observed.

Whoever undertakes this mode of therapy should acquaint himself with both the theory and the technic. It is not to be undertaken lightly nor carelessly, and general instructions are no more than points of the compass; the path to be followed is determined by a conscientious study of the individual patient and his reaction to each injection.

Autogenous vaccines should always be used, if possible, both because the organism concerned is certainly established and because the antibodies produced are so specific that even other strains of the same organism do not give as good results. Stock vaccines may be used where the nature of the organism is determined, and the delay in the preparation of the vaccines may prejudice the patient's condition, or where a culture may not be obtained and the clinical features of the case make the diagnosis fairly certain, as, for example, that over 50 per cent of the cases of pyelitis are due to the bacillus coli and that the urine is characteristically acid in these cases, might warrant the use of stock colon vaccines. The number of injections varies with the response, from three or four to a dozen or more. The best intervals are about four days.

It might at first seem irrational to expect a body already overwhelmed with poisons to react favorably to the imposition of more, but the formation of antibodies would seem to be a local reaction at the site of the inoculation and occurs when the tissues at the site of the original infection can no longer respond; and these antibodies escaping into the circulating fluids effect in their own particular way a decrease of the toxins.

Operation.—The operative procedures one must have recourse to in a certain number of these cases can be referred to here only in the briefest manner; for detail reference must be made to a surgical treatise. Before operation is undertaken an earnest effort to determine the nature of the infecting organism should be made by cultures of the urine taken from either ureter, blood cultures, or cultures from probable sites of primary infection. If bacillus coli is determined, the case is in quite another category from those due to such pyogenic organisms as Streptococci pyogenes and staphylococci, for the tendency of the latter is to abscess formation, of the former to diffuse inflammation.

Clinically, the one striking difference in these two groups is the absence of profound toxemia and prostration in the colon group, though chills may initiate the attack, the fever run high, and local signs be acute.

In the colon cases a surgeon is loath to operate, unless progressive toxemia occurs or the case be exceptionally fulminating in character, and then he is prepared to be more conservative and prefer decapsulation, incision and drainage of infected areas to an immediate nephrectomy.

In the pyogenic cases, if an uncomplicated perinephritic abscess exists, incision and drainage are indicated; but with involvement of the kidney substance itself the question of operation resolves itself into a choice between nephrotomy and nephrectomy. If nephrectomy is to be done it is imperative to know the functional ability and structural integrity of the remaining kidney; and, as the decision between the two procedures may not be settled until the kidney is exposed in the course of the operation, functional tests should be made in each instance. The three constituents of the urine to be determined are water, salts, and nitrogen.

Although in certain forms of nephritis sodium chlorid is discriminated against, particularly, by the kidney (the determination is not difficult), still it is the first and last functions that concern us here.

Catheterism of the ureters readily determines the output in a given time of one and the other kidney in terms of water and nitrogen, and, moreover, affords a study of the exudate from each kidney.

Innumerable methods have been employed to determine the question of functional integrity, physical methods such as cryoscopy, electrical conductivity of the urine, the eliminations of drugs: potassium iodid, the salicylates, phloridzin, dyes—such as methylene blue, rosanilin, etc. The chief functional tests now used are the chemical examination of the blood for nitrogen and salt retention and the output through the kidneys (or kidney) of phenolsulphonephthalein or indigo carmine. These tests with the most thorough examination of the urine, particularly when the product of each kidney is compared with the product of its mate (synchronous bilateral ureter catheterism) with bacteriological findings serve to afford very accurate conclusions as to renal function.

Complete destruction of a kidney or serious involvement of the structure, multiple abscesses, would indicate immediate nephrectomy. With a lesser degree of impairment of one kidney and integrity of the other it is a nice question, determined more or less by the localization of the process, whether it is better to do a nephrotomy and drain, hoping to retain a goodly portion of the damaged kidney; and, failing of prompt healing, do a nephrectomy later; or, whether it is better to do a nephrectomy at once, fearing that the retention of a damaged kidney may imperil the other before nephrotomy has demonstrated its efficiency; or the secondary nephrectomy is done. In the light of the compensatory growth of the remaining kidney, and its capacity for meeting the excretory needs, the tendency is to take no chance of imperiling the sound kidney and favor immediate nephrectomy.

If both kidneys are involved the question of nephrotomy or nephrectomy must depend on the relative involvement of the two kidneys. A suppurative process may still be compatible with a high degree of functional capacity; and the excision of a kidney that has no function and simply pours its bacteria and toxins on the relatively good kidney is certainly indicated. On the other hand, if both kidneys are badly impaired, the excision of one will precipitate a final issue, to which the operation itself may be the determining factor.

COMPLICATIONS

The pyelonephritis may be associated with, indeed due to, tuberculosis of the kidney (see Tuberculous Kidney) or renal calculus (see Nephrolithiasis), and may therefore introduce the problems of these diseases.

Cystitis.—Cystitis may be due to a pyelonephritis of hematogenous origin. The treatment of the cystitis, then, is a most serious consideration, involving the removal of the exciting cause, such as stone, enlarged prostate, changing the reaction of the urine, use of urotropin, bladder irrigations, and the use of vaccines.

Retention of Urine.—This may occur, most commonly due to hypertrophied prostate, and to acute and chronic prostatitis, and, as has been said earlier in this article, powerfully favors the advance of infection. Prostatectomy or other treatment of the prostatic condition is indicated.

Suppression of Urine.—Suppression is an ominous symptom, and demands all the efforts directed at this condition under other circumstances. To suppression will probably succeed uremia, in the combating of which the measures usually pursued (see Nephritis) are attempted as a forlorn hope. The infection from being local may become general and a true *septicemia* follow. Here the measures applied in any acute infection obtain: fresh air, sufficiency of easily digestible food, cold water to control temperature, if excessive; heat to the skin and internally when chills occur; the use of vaccines, in the smaller doses more frequently administered. When such a condition prevails our efforts are to make the patient comfortable, in which we may be successful, to combat the infection by whatsoever measure holds out any promise, but at the present moment unfortunately with very little likelihood of success.

Peritonitis.—Peritonitis is a rare accident due to rupture of an abscess into the peritoneum, and must be dealt with surgically.

Cardiac Hypertrophy and Hypertension.—This is an expression of interstitial changes in the kidney, and is to be considered in the same light as when occurring in chronic interstitial nephritis—support for the heart when the compensation breaks, and study of the hypertension to determine at what point this increased blood-pressure constitutes an optimum for the individual patient.

Gastro-intestinal Symptoms.—These are sometimes a source of great discomfort to the patient, and are often the manifestation of uremia.

Gastric sedatives, bismuth, soda, cerium oxalate, gastric lavage, cracked ice, cocain, or even morphin, in the worst cases; local measures, heat to epigastrium, or cold, or counterirritation are all to be tried. Diarrhea must be controlled, if excessive, by bismuth or opium, but it must not be forgotten that uremic diarrheas are probably of purposeful significance in the elimination of poisons; in constipation mild vegetable cathartics and colon irrigations are depended upon.

PROPHYLAXIS

Prophylactic treatment begins with all those rules of hygiene, diet, and exercise that make for a healthy body. Next, it takes cognizance of possible causes of suppuration of the kidney and endeavors to remove them or render them less likely to become operative.

Recognizing the acute infectious diseases as possibly excitant of the process by hematogenous infection, a rational treatment of the disease concerned, an abundance of water, care of the other emunctories, especial attention to local lesions in the course of the disease, such as tonsillitis, otitis, adenitis, or abscesses, and frequent careful examination of urine lessen the likelihood of pyelonephritis. The administration of urotropin in the latter days of a typhoid fever is a preventive measure of importance. Considering the possible sources of an ascending infection, suppurative processes or inflammations or mechanical impediments in the urinary tract should receive attention. These include gonorrheal urethritis, cystitis from any cause, ureteral, renal or vesical calculus, hypertrophied prostate, pelvic masses that might be obstructive, a bacilluria without obvious signs of inflammation, movable kidney which by kinking causes partial obstruction and stasis, or gives rise to Dietl's crises, conditions favoring infection.

Instructions should be given to mother and nurse in the light of the frequent infection of female infants and children to wipe excretions away from rather than toward the vulva, and observe strict cleanliness of the parts and clothes.

Appreciating the road direct by the lymphatics, and that infection through the blood may come from the same source, the bowel should receive attention, constipation be avoided, and bacilluria, usually from bacillus coli, during intestinal disease, be early detected and treated by urotropin, abundance of water, or by alkalis.

Finally, attention must be given to diseases of the kidney itself, to which pyelitis and pyelonephritis are common, to eradicate them as far as possible, namely, nephrolithiasis, tuberculous kidney, movable kidney, and hydronephrosis.

Surgical cleanliness must be observed on the occasion of every necessary instrumentation of the genito-urinary tract.

REFERENCES

Rowntree and Geraghty. Journ. Pharmacol. & Exper. Therap., i, 6, 1910.

Wildholz. Quoted from Kretschener, Colon Bacillus Pyelitis, Int. Med. Journ., Feb., 1915.

CHAPTER XXVII

HYDRONEPHROSIS

Frank Sherman Meara and Alfred Townsend Osgood

Hydronephrosis may be either congenital or acquired; it is with the latter that we are especially concerned in this chapter.

Acquired hydronephrosis is the result of an obstruction, partial or complete, to the flow of urine anywhere along the course of the urinary passages. It is more likely to be bilateral, if the cause is operative in the bladder, prostate, or urethra. Pelvic tumors, growths, or peritonitis may involve one or both ureters, depending on their extent. A movable kidney, especially if the upper portion of the ureter is fixed, by causing a kinking of the ureter, is another potent cause. It must be remembered, too, that infection of the pelvis or ureter, without other cause for stenosis, may be responsible for hydronephrosis. Pelvic tumors, inflammation, and movable kidney make the condition more frequent in women than in men. Perhaps the most common cause is renal calculus, which causes stenosis of the ureter, on the one hand by impaction and on the other by cicatricial contraction secondary to trauma and ulceration caused by the passage of the stone. Stone and movable kidney are much more likely to induce unilateral lesion.

Four results are likely to ensue upon the development of a hydronephrosis, each of which has both local and remote results:

First, the tumor itself (and this is not always large, but often too small to make out), which, from its size, may interfere with the relationship of the surrounding organs, and with their function.

Second, the huge collection of water in the pelvis induces atrophic changes in the parenchyma of the kidney, even to the point of complete destruction, thus depriving the body of the function of that organ and throwing all the work on the other kidney.

Thirdly, if Castaigne and Rathery were correct in their insistence, there is always a lesion of the opposite kidney, which, from their clinical and pathological observations, and from their animal experimentations, they attribute to the toxic effects of the products of cell destruction in the nephrotic kidney, and they assert that the serum of an animal on whom a ligature of the ureter has been practiced acquires nephrotoxic properties. Thus a chronic nephritis is induced in the opposite kidney, with terminal uremia. Although their theories have not been universally accepted the observation that the opposite kidney may be affected has been frequently made and constitutes a potent reason for operative interference.

Fourth, these kidneys are prone to infection, and a pyelonephrosis or pyonephrosis set up with consequent general toxemia or septicemia.[1]

Hydronephrosis may be permanent or intermittent, and it is the latter type that affords a very interesting clinical picture. It is almost invariably unilateral, and is due to the kinking of the ureter of a movable kidney, or to an impacted calculus, the occlusion so caused being overcome by the collecting urine. Braasch believes this type to have an anatomical basis, due to congenital conditions and finds the average age at onset of symptoms to be twenty-one years.

The characteristic feature of intermittent hydronephrosis is the formation of a huge tumor, which disappears with the simultaneous passage of a very large amount of urine; however, it must be kept in mind that a hydronephrosis is not always huge, not always even a manifest tumor. With the accumulation there may be pain, at times excruciating, and relief on disappearance of the mass.

In an interesting case I had for some time under observation the collection would be accompanied by severe pains, like renal colic, and gradually disappear, the huge collection remaining, at times, for weeks, then, without warning, emptying itself, either to remain empty for a long period or to reaccumulate at once. It would seem probable that the degree of pain would depend somewhat upon the rapidity of accumulation and be the more severe in a kidney which had not yet undergone much change.

A tumor is by no means constant and much damage may be done to the kidney before collections sufficient to afford a palpable tumor occur. Cabot has made a strong plea for early diagnosis, recalling that the symptomatology may be that of renal colic or pyelitis, even when no stone is present or the urine with pus and blood, suggesting infection, is sterile.

Pyelography helps greatly in the appreciation of these cases and, indeed, in the vast majority of cases diagnosis is determined only by catheterism of ureter and pyelography. Operation is early indicated, before irreparable injury to the kidney parenchyma ensues.

Treatment.—Bilateral congenital hydronephrosis is incompatible with life. Unilateral congenital hydronephrosis will compel surgical intervention.

---

[1] C. Fenger noted the abnormal arrangement of renal arteries as one of the causes of hydronephrosis. Mayo, Braasch and MacCarty found an anomalous arrangement of renal vessels at the point of ureteral obstruction in twenty of twenty-seven cases of hydronephrosis.—Editor.

In the case of acquired hydronephrosis the question to be decided is whether operation is or is not indicated.

Patients have gone on for years with no apparent ill effects from the condition until the parenchyma of the kidney has been destroyed, and this knowledge often persuades to conservative treatment, and, in the intermittent cases, a failure to refill and a spontaneous cure has been known to occur, but the danger of infection ascending or descending is always present, and the more remote possibility of rupture from traumatism.

The rational procedure is to set about to determine the site and nature of the obstruction, its degree, the integrity of the affected kidney, and the presence or absence of any lesion of the other. This involves exploration of the urethra, cystoscopy to determine obstructive conditions in the bladder, catheterism of ureters, to determine the degree of stenosis, its site, perhaps its character, as the use of wax tip in searching for stone, the collection of urine from either side to determine rate of flow and character of urine, thus discovering the amount of functional impairment of the affected kidney, and evidences of nephritis in the other. X-ray plates should be secured to detect a possible stone, the distention determined by pyelographic methods and thorough abdominal, rectal, or vaginal examinations made.

It cannot be too emphatically insisted that no operation should be undertaken on a kidney without knowledge of the functional status of the other by a study of the blood chemistry, phthalein output, and, by ureteral catheterism, the urea output and phthalein elimination of the residual kidney.

I am convinced that, with the perfection of technic of to-day in this field of surgery, an operation is always indicated, which, exploratory at first, proceeds on the basis of its findings. If a stone is found, and the previous examination of the urine from that side or the appearance of the kidney warrants the assumption of a functioning organ, the stone is to be removed and the kidney saved; if a movable kidney is discovered and a restoration to its normal position promises to relieve the condition, the kidney being still functioning, this operation is indicated. Relief of constriction or plastic work on the ureter may be indicated.

If the kidney has been practically destroyed, or the obstruction cannot be removed, or the kidney is infected, it remains but a menace to the other kidney and the life of the patient, and nephrectomy should be done, or nephrotomy, followed by a secondary nephrectomy, depending on the findings at the operation.

For the attacks of pain, the same local and drug measures may be used as in renal colic (see Nephrolithiasis).

In some instances the patients contrive to find a position that effects a release of the accumulation, and at times manipulation of the tumor mass, in an effort to elevate it or gently massage it, does the same; but extreme caution must be used in such efforts.

The accepted procedures are: (1) Catheterism of the ureter, which when successful, of course, leads to direct evacuation of the retained urine. (2) Operation under local anesthesia exposing the kidney in the loin and draining the pelvis directly through the parenchyma. Aspiration is mentioned only to condemn the procedure.

After the attack of pain, if operation has been refused, effort may be made by seeking various positions or by the use of binders and belts to effect an unkinking of the ureter. If infection has occurred and the hydronephrosis is converted into a pyonephrosis, operation is doubly imperative; if refused or not feasible the same line of treatment is carried out as suggested under similar circumstances when treating pyelitis. Cystitis is to be treated, as under other circumstances, by local applications, drugs, and irrigations.

The other kidney may not go scot free, and, as has been said, it is maintained by good authority that the presence of the hydronephrotic kidney, not its absence, imperils the other kidney, and a sclerotic condition may ensue, with uremia as the result. Our task is to appreciate the involvement of the second kidney before this accident occurs, and take such measures to reduce its task by dietetic and hygienic precautions as in any case of chronic nephritis. The development of uremia entails the usual procedures for this condition (see Uremia), but with the knowledge that the fight is that of a forlorn hope, and the appreciation that the best we can do is to lessen suffering by controlling the gastric distress and preventing the onset of convulsive seizures, more distressing to family and friends than to the patient at this stage.

REFERENCES

Beer. Experimental Study of the Effects of Ureteral Obstruction on Kidney Function and Structure, Am. Journ. Med. Sc., June, 1912.
Braasch. The Clinical Diagnosis of Hydronephrosis, Int. Med. Journ., Nov., 1914.
Cabot. The Diagnosis and Indications for Operation in Early Hydronephrosis, Journ. Am. Med. Ass., Jan. 4, 1913.
Mayo, Braasch and MacCarty. Ibid., lii, 1383, 1901.

CHAPTER XXVIII

NEPHROLITHIASIS

Frank Sherman Meara and Alfred Townsend Osgood

Nephrolithiasis is a condition which, in one or more of its manifestations, must be met repeatedly by every general practitioner of medicine; a condition in the consideration and treatment of which the internist, the surgeon, the genito-urinary specialist, and the roentgenologist are called upon for their best endeavor.

The gravity of the affection depends very largely on the presence or absence of infection. As long as the patient is free from infection his symptoms are referable only to the mechanical effects of the stone, the pain incident upon its movement through the tract, the disturbance of function induced by the irritation, and the obstruction ensuing upon its impaction; but, when infection obtains, the case takes on a new and more serious aspect. The trauma, stagnation, and interference with function attributable to the mechanical effects of the calculus render the field fertile to the seeds of infection, which may arrive at the point of least resistance either from below, by way of the genito-urinary tract, the so-called ascending infection, or from above, by way of the renal circulation, the so-called descending or hematogenous infection.

While in the vast majority the stone is primary and the infection secondary, there is still a group of cases in which a previous infection of the urinary passages has furnished the conditions under which stone formation may occur.

While it is possible for calculi to be present in the form of sand or of the coarser gravel or even at times as a considerable stone, and give rise to no symptoms whatsoever; and, while such symptoms as are present may be of a lesser character, giving rise to discomfort and dull backache, which may be attributed to lumbago or neuralgia; or, finally, while the infection, as a pyelitis, a cystitis, or a pyelonephritis, may dominate the picture, nevertheless our earliest introduction to the condition and our most common experience with it are, as a rule, on the occasion of one of those violent crises of pain termed a renal colic.

The pain may be of any grade of severity; as a rule, very decided, often agonizing, beginning in the lumbar region and radiating in a characteristic way around the side of the abdomen to the groin, into the scrotum and testicle, or into the labia majora in the female, often into the penis, down the inner side of the thigh, and at times into the rectum and perineum. Nausea and vomiting are common accompaniments of the pain, as they are of severe attacks of hepatic colic. The pulse becomes rapid, the patient is prostrated, or may go into collapse. Not infrequently a chill occurs and fever follows, and this, too, in cases not recognized as septic. Where infection has taken place the chills and fever may be so severe and frequent as to give rise to the diagnosis of malarial paroxysms. Frequency of micturition may or may not take place, and, according to Young, depends on the position of the stone, those which are low down in the ureter, intramural, or projecting into the bladder, exciting desire to urinate. Sometimes suppression follows and may continue for hours or days, due more often to cessation of function from irritation than from blocking. The attack may be very brief or last for hours, leaving a dull ache as an aftermath, while the urine voided shows, from the red and white cells it contains, the trauma suffered by the passages.

It seems advisable to take up the treatment much as the demands are made on us in practice rather than in the more formal manner of a treatise.

TREATMENT

Renal Colic

Pain, and pain alone, is the consideration of the moment. The demand for relief is imperative. All of the immediate symptoms are due to the pain and disappear with the pain. With the first premonition of an attack or in attacks of lesser severity relief may be afforded by the use of heat in the shape of a hot bath. It is one of the procedures the patient himself may initiate, awaiting the advent of the physician. It is possible that the attack may be aborted or greatly ameliorated by this means. The patient enters a tub containing enough water to submerge the trunk and limbs. The temperature of the water on entering should be about 100° F. and gradually increased to 110°F. to 115° F., depending upon the patient's sensitiveness to heat. Cold applications are made to the head during the bath. The bath is continued for five to fifteen minutes, being interrupted on the occasion of any feeling of faintness. The effects may well be continued by rolling the patient in two or three dry blankets which are made to inclose the body snugly, while cold is still applied to the head. In the pack the patient often sweats profusely. When taken from the bath he should be rubbed dry, given an alcohol rub, but the body still kept warm. The rationale of this procedure depends on the effect of warmth on the action of muscle tissue.

Such experimental data as we have at hand point to a diminution of tone of both striated and smooth muscle under these conditions. Such an effect would mean a relaxation of spasm, and thus experiment has confirmed experience, for the relaxing effect of the warm bath in renal colic has long been accepted by practitioners of medicine.

Local Application of Heat.—When from the circumstances of the environment, a hot bath is not obtainable, or from the severity of the pain, or the weakness of the patient, it is not feasible, heat applied locally may afford some measure of relief.

Hot Fomentations.—The most rapid and efficacious method of applying heat is in the shape of cloths wrung out of hot water. Two or three pieces of thick flannel cut to the size of the site of application are saturated with boiling water, as they lie in a crash towel or other container, the ends of the towel being twisted in opposite directions to squeeze out the excess of water from the flannels it contains. Sticks attached to the ends of the towel facilitate the process. The site of application is over the lumbar region of the affected side or over the site of greatest pain. The skin is smeared with oil or vaselin, the degree of heat of the compresses tested to avoid a burn, the cloths then laid on smoothly, and dry flannel placed over them. The fomentation is left on for five or ten minutes, then another applied until three or four have been done. If the procedure has been efficacious for the time, it may be repeated on a recurrence or aggravation of the pain.

Poultices.—While the poultice is not so readily prepared as the fomentation, it has the advantage of affording heat for a longer time and sparing the patient the disturbance of repeated applications. Perhaps the best material for the poultice is flaxseed meal.

The Hot-water Bag or Bottle.—In no form can heat be more readily applied; but the application cannot be so intimate throughout the area to be considered as with other methods. Care must be taken that there is no drop or leak, and sufficient flannel intervene between bag and skin to regulate the temperature. The bag should not be more than half full, the air expressed, by squeezing the water to the nozzle level, and the cap screwed firmly on. In this way the bag becomes more pliable and makes more easy its application and retention.

Hot Douches.—While affording another means of applying heat locally, it is rarely attainable except in institutions, and not applicable in the severe cases.

These measures, like other measures of physical therapy, are our best heritage from empiricism, and the belief in their efficacy has been fostered much more constantly by the laity than by the profession. Just at present, to my mind fortunately, physical therapy is enjoying a renaissance. Much work has been done to analyze the effects of these physical agents, yet it is at the best but pioneer work.

As to the rationale of the application of heat locally in the condition under discussion, I cannot do better than quote Matthes' summation of our knowledge of such effects, namely, "One can actually influence the distribution of blood hydrotherapeutically (1) through the local application of heat or cold, which operates directly at the site of application, upon the vessels or reflexly; or (2) through the reaction upon other vascular provinces exerted hemodynamically or reflexly. We can further assert that we are in a condition to render the skin richer or poorer in blood and that we can in this way exert a considerable influence on the circulation of the internal organs. Unfortunately this influence on the circulation of the internal organs is very incompletely known in its details."

It is possible, then, that a relief of congestion or inflammation induced by the trauma may be afforded in this manner, and that the relaxing effect upon the ureteral structures occurs from the local application of heat as it is believed ensues when the application is general, as in the bath.

Cold.—Some cases are said to find more relief from the application of cold than heat, but I think they are relatively rare. The best mode of application is the ice-bag. I prefer the circular one. Use small pieces of ice, enough to cover the bottom, water enough to cover the ice, and express the air to render the bag supple and easy of application. Put gauze or muslin between bag and skin.

Enteroclysis.—Warm water in the bowel may afford some relief of pain; it is best used in the form of an irrigation. The temperature of the water should be 105° F. to 120° F., using two to eight gallons of plain water. This is introduced into a fountain syringe or douche bag, held 3 to 5 feet above the level of the bed. A two-way tube should be used, the most easily improvised being two catheters, one of a slightly smaller caliber than the other and protruding slightly beyond the other, the smaller for the introduction of water and the larger for its discharge and that of the small solid particles that may accompany it. These may be kept together by silk ligatures, or may be sewed together. The tubes should be well oiled before introducing, and no force used in the procedure. If obstruction is met, wait until any spasm provoked has ceased before continuing. Allow a little water to flow, thus distending the bowel ahead of the tube and facilitating its passage. Pass the tubes in 6 to 9 inches. A Kemp tube, especially devised for the process, may be used. A relaxing effect on the spasm of the ureter by the water in the contiguous structure probably exerted reflexly may be assumed. The diuresis provoked by the procedure may also be helpful in dislodging the calculus.

Warm Drinks.—Warm milk, barley water, Vichy, or lemonade may be given with benefit. Probably a relaxing effect is induced by warm water taken internally, as it is when locally applied; at least, fall in blood-pressure attributable to lowered vascular tone has been observed after the ingestion of warm drinks.

Posture.—There are certain postures in which the patient may find the greatest relief. He will most certainly seek such, and the position should be respected as much as possible by the physician in his procedures. Occasionally inversion of the patient is efficacious.

The above measures are applicable only in the attacks of lesser severity, or when the severity of the attack has been lessened by other measures. In many cases, perhaps in most cases, of renal colic the demand for relief is so urgent that immediate recourse must be had to drugs.

Drugs.—There is no hesitancy in the choice of drugs under these circumstances. Morphin stands preëminent. It must be given hypodermically, down into a muscle for a more rapid effect, and it must be given in sufficient quantity. Not merely age and weight, but the pathological condition treated determines the dosage of morphin, and severe pain requires large doses. Indeed, in the cruelly punishing attacks of colic, amounts of morphin that trespass closely on the danger line under other circumstances may neither relieve nor manifest the usual toxic effects. In the adult, if the colic demands morphin, gr. ¼ (0.015 gm.) should be the beginning dose, and, if some result is not seen in a quarter of an hour, another dose of gr. ⅛ (0.008 gm.) may be given, which, in turn, may be repeated in the same length of time.

Lesser doses but delay the effect, the patient is nagged by the frequency of administration, and the physician left uncertain as to what his next dosage should be when several small ones have already been administered. The same unsatisfactory train of events ensues if attempts are made in the severe cases to begin administration by mouth or rectum, and, as our doses are usually larger when given in this manner, we are left uncertain, when we feel compelled to give a hypodermic, as to just how much has already been absorbed and what our hypodermic dose shall be.

Chloroform.—Another drug that occupies a place second only to morphin, and, in exceptional cases, first, is chloroform. In the most violent attacks morphin may not afford relief in doses that the practitioner is willing to give.

I have given, in a case of hepatic colic, gr. ¾ (0.045 gm.) of morphin in a very short space of time without effect. I did not care to go beyond that. In these cases a few whiffs of chloroform will afford relief, but often not before the patient is mildly anesthetized. When the intensity of the spasm has thus been broken up, the morphin previously administered becomes operative and is efficient in preventing the recurrence of the attack. In these very severe cases it is often well to give the chloroform at once, and then, with any premonition of recurrence, give morphin, gr. ⅙ (0.01 gm.). The chloroform should not be pushed or continued for any length of time, but enough given to relieve the spasm. Another indication for chloroform in preference to morphin is in those cases which have a decided idiosyncrasy for morphin.

Finally, one feels warranted in giving patients who are prone to such attacks and likely to be taken when remote from assistance the advice given by Lauder Brunton to patients suffering from angina pectoris, namely, to place a sponge in a small bottle with a wide neck, and, on the occasion of an attack, wet this sponge in chloroform and inhale. As the patient drowses off, the bottle will fall from his hand, and no harm from overdose can occur.

Its effect may be permanent, owing to the discharge of the calculus during the period of relaxation or to the relief of the pressure behind the obstruction, the relaxation permitting of the passage of urine by the impacted calculus; or its effects may be evanescent owing to a recurrence of the original conditions giving rise to the attack. In these latter cases the addition of the morphin is needed.

The usually accepted view of the cause of the colic is the spasm set up by the irritation induced by the calculus. Bevan, however, is confident that the passage of the stone through the ureter itself does not cause any pain at all, but that "the cause of a renal colic is a greatly increased intrarenal tension due to a plugging of the ureter"; and, while the former view is the simpler, still the rapid disappearance of the attack with relief over long intervals, though the stone is shown by the X-ray to remain in situ, the passage of stones of considerable size and roughness without pain, and the attacks of colic induced by blood-clots or other soft substances add weight to Bevan's view.

Atropin.—Because of the striking effect of this drug on all unstriated muscle substance, whose function it diminishes, it is used when these structures are in a condition of spasm. The best way to administer atropin is hypodermically, and a sufficiency used to effect the purpose: gr. 1/100, gr. 1/75, or gr. 1/50 (0.0006 to 0.0012 gm.). It may be used in combination with morphin, gr. 1/150 to gr. 1/75 (0.00045 to 0.0009 gm.), thus bringing the sedative effect of the two drugs to bear at once. Each tends to bring relief, though the modus operandi is not identical. Another advantage of the combination of the two drugs lies in the fact that the nausea and vomiting, which in many patients are the inevitable sequelæ of morphin administration, are greatly ameliorated or quite prevented by the atropin. Here, again, one must be assured that the patient has never shown marked idiosyncrasies for the members of the belladonna group.

Chloral.—When the pain is less severe, or has in a measure been controlled, chloral, on account of its sedative effect on all nerve structures, may break the reflex between sensory irritation and muscular response in the ureter and thus bring relief. It may be given by the mouth in doses of gr. xv to xx (1.0 to 1.3 gm.), or by rectum in an ounce or two of warm milk, in double the dose, gr. xxx (2.0 gm.).

I refrain from mentioning the drugs of lesser activity classed as sedatives, because their usage is not followed by satisfactory results in such decided spasm as renal colic, and but defers the measures of real value that have just been cited.

Treatment of Other Symptoms Than Pain during the Attack—*Nausea and Vomiting.*—Nausea and vomiting are so intimately connected with the pain, so secondary in importance to the pain, in the minds of both patient and physician, and so promptly disappear with the pain that our attentions are directed entirely toward the latter. The treatment of the nausea and vomiting itself, except through relief of the colic, would be worse than useless. Those of us who seek for the protective, curative, or compensatory significance of the symptoms in disease will find it in the relaxation of the spasm induced by the vomiting that facilitates the discharge of the offending calculus.

Collapse.—Prostration and collapse may occur in very severe attacks and in patients already weakened. It should be the same as for collapse in other conditions: heat to the extremities, a hot saline irrigation at 110° F., to which a cup of strong black coffee may well be added, diffusible cardiac stimulants, like ammonia, the vasomotor stimulants, adrenalin, ♏ xv to xx (1 to 1.33 c.c.), into the muscle, caffein, as a soluble salt, benzoate or salicylate, gr. v (0.3 gm.), or camphor, gr. v (0.3 gm.), in 10 per cent to 20 per cent solution in sterile oil, or, if the heart is weak, strophanthin, Merck, gr. 1/120 to gr. 1/60 (0.0005 to 0.001 gm.). All these may be given hypodermically, or, better, intramuscularly.

Chill and Fever.—This may occur with the colic when there is no infection. It passes with the spasm and needs no special treatment, unless out of proportion to the pain, when heat to the body during the chill and cool sponge baths during the fever may be given. Recurring chills and fever occur with infection and have been considered under pyelonephritis.

Suppression of Urine.—The simple act of catheterism determines whether we are dealing with retention or suppression. The first twenty-four hours gives us little alarm, as transitory anureses lasting twelve to twenty-four hours may occur after an attack of colic, as it may after an operative procedure on the kidney or other abdominal organs, but when it runs beyond the first twenty-four hours the possibilities of a persistent anuresis are greatly increased. There is a curious tolerance, a freedom from any symptoms of distress, that lasts five or six days, that may lure the careless into a sense of security; then come on a feeling of fatigue, a restlessness, sleeplessness, and digestive disturbances that betoken the earliest toxic effects of retained products, then follows a uremia, the patient sinking into a coma in which he dies; rarely are convulsive seizures present. The ureter may be catheterized in these cases, passing beyond the stone and, by relieving the tension, remove factors determining suppression. I have seen considerable accumulation of ureanitrogen in the blood, resulting from the shock of passing a stone, clear up in two or three days.

The mortality of non-operated cases is large, about 70 per cent.

Vigorous treatment in the early stages is indicated, and the earliest signs of poisoning should hasten any surgical procedure of promise. In suppression decapsulation is effective in many cases when operated early.

That many of the kidneys of nephrolithiasis are in a condition of chronic congestion we know, and, appreciating the possibility of an increase of congestion as contributing to the anuresis, we apply cups over the kidney region, wet or dry, or hot fomentations. We further relieve the congestion and enhance a vicarious function by inducing vigorous catharsis, by the use of salines and diaphoresis, by the use of the hot pack or hot-air bath, or, in the absence of facilities for carrying out these measures, pilocarpin hydrochlorid may be given in doses of gr. 1/10 to gr. 1/5 (0.006 to 0.01 gm.).

Failure of response of the kidneys to these efforts at relief leads us to stimulate their function by bringing to them normal products of excretion such as water and sodium chlorid, best administered, the water by mouth or with salt as a physiological salt solution by the rectum by means of colonic irrigation, using 2 to 8 gallons at 105° F. to 120° F. One may have recourse even to hypodermoclysis and infusion. With the onset of uremia the infusion may be preceded with benefit by venesection.

Drugs.—Drugs, as diuretics, may be used, my preference being for the theobromin-sodium-salicylate or diuretin in doses of gr. x to gr. xv (0.6 to 1.0 gm.) three or four times a day, or some other member of the same series, such as theocin, gr. iii to v (0.2 to 0.3 gm), t. i. d., or, in the case of weak heart, caffein in the same doses. Alkaline salts may prove useful, the citrate, bicarbonate, or acetate of potash or the acid potassium tartrate (cream of tartar) in dose of gr. xv to xxx (1.0 to 2.0 gm.) every two hours, singly or in combination.

Lemonade or imperial drink (cream of tartar ℥ss to O iii (15 gm. to 1,500 c.c.) of boiling water, and lemon peel and sugar to the taste) may be given ad libitum. Should the major accidents of uremia, convulsions, occur they are to be treated as in the uremias of nephritis (see Nephritis).

Death from suppression usually occurs on the tenth or eleventh day.

After an attack the patient should remain in bed until the ache and soreness have passed and the evidences in the urine of irritation of the stone have ceased.

When infection occurs the picture changes, and we have to deal with pyelitis and pyelonephritis (see Pyelitis).

LITHIASIS

When an attack of renal colic has occurred or the presence of sand, gravel, or calculus has evidenced that a state of lithiasis exists, certain precautions must be observed to avoid an aggravation of the condition or prevent the onset of or recurrence of an attack.

The patient should lead a quiet life with avoidance of excesses of any kind. Violent physical effort or sudden exertion may precipitate an attack of colic. This does not mean that a sedentary life should be encouraged, for this is a factor rather in the production of the condition.

Exercise.—Exercise is important in that a normal functioning of the muscles encourages a normal metabolism in these structures. It must be light in those in whom colic has occurred, such as walking and calisthenics and massage; it may take on more vigorous forms in those in whom sand or gravel alone have appeared, while it may be said that, other things being equal, the younger the subject the more active may be the exercise.

The links in the chain of events that give rise to calculi are not all of them clearly discernible. Our present theories assume that in the intermediary stages of the catabolism of certain organic substances, members of the purin group, or in the play of physicochemical processes in the disposition of certain inorganic salts, such as the phosphates, errors creep in, errors which are determined either by heredity or acquisition; and, further, that damage to the kidney, either a trauma by salts precipitated in its tissue or bacterial infection, plays its part.

It is on this assumption that we order our therapy, to improve the functions of the body by wholesome living, avoidance of excesses, exercise, fresh air, baths, sufficiency of sleep, and proper correlation of work and rest, aiming to lessen the tendency to errors in metabolism, inherited or acquired. We so order our diet as to burden as little as possible the tissues with the substances difficult of catabolism; that we avoid or ameliorate infection, and encourage elimination.

Diet.—Our diet is determined by the nature of the error in metabolism and that in turn by the examination of the stone or gravel; hence the importance of examining each passage of urine to obtain the calculus that may pass. The vast majority of calculi belong to one of three groups: uric acid, oxalates, or phosphates. Oxalates and uric acid form one group, found in acid urines, while the phosphates, found in alkaline urine, form the second. The phosphatic calculi are as a rule secondary to infection and urinary fermentation, though they may, like the first group, be primary. Kahn and Rosenbloom find that the vast majority of renal calculi are composed of oxalate of lime and, though uric acid and urates are found in nearly all, it is the oxalates that compose the main portion.

Two fundamental principles are to be observed in all cases: (1) simplicity of diet, that is, avoidance of rich food, multiplicity of courses and foods in a course, and (2) sufficiency and only sufficiency, that is, for a man of ordinary activities about 40 calories per kilo, or 2,800 to 3,000 calories for the man of average weight.

When the stone or gravel is composed of uric acid, meats should be cut down to small quantities, and especially those containing much cellular material like sweetbread, kidney, liver, or brain; all smoked, spiced, and highly seasoned food and rich pastry should be forbidden. Fats and sugars should be given in moderation, and the amounts of vegetables, fruit, and milk increased.

When the stone is composed of oxalates those foods that contain much oxalic acid or substances from which oxalates may be derived should be avoided. Most vegetables (except the legumes, for example, peas and beans) are interdicted, especially spinach and rhubarb, on account of the oxalates they contain and the large amount of calcium in most vegetables, which renders the oxalates less soluble. Figs, strawberries, apples, and pears should be included in this list of articles forbidden. On the other hand, meats are to be allowed more freely, except the glands, thymus, liver, etc., named above, and according to Klemperer, gelatin, as all these substances are said to give rise to oxalic acid in the process of metabolism.

When the stone is composed of phosphates the vegetable diet should be decreased and the meat increased.

In many cases the nature of the stone is unknown or it proves to be mixed and, as Klemperer has wisely said, "the prophylaxis of renal calculus lies in the mixed diet dictated by nature," and, indeed, while we seek to be guided by the data of scientific experimentation and research, our deduction should not lead us too far from the road of experience, and we should feel that until our information is very definite it is safer to steer the middle course and not depart too widely from the diet nature has laid down for man.

Alcohol.—Alcohol is to be strictly forbidden. Its ingestion interferes with, not enhances, the metabolic processes.

Water.—That concentration of the urine, that is, oliguresis, facilitates the precipitation of the salts within the kidney substance is a part of the theory of calculus formation, and for this reason as well as for the purpose of flushing out mechanically the small crystals formed before concretion can ensue, water is given in abundance. The quantity should amount to 2½ to 3 quarts, which usually affords 48 to 64 ounces of urine. The water intake should be frequent, every two hours, or at mealtime, between meals, and on retiring.

Alkaline Waters.—It is often advised to give alkalis with the idea that they have a solvent effect on the stone formed, which seems to me extremely dubious, but they do act as diuretics and do diminish the acidity of the urine, a reaction which favors the formation of urates.

If, as the analyses of Kahn and Rosenbloom would seem to show, oxalate of calcium plays so large a rôle and uric acid and its salts so small a one in the formation of renal calculi, their contention that alkalis are contraindicated and advice to render the urine acid would seem logical. One would use benzoates or salicylates for such a purpose in doses sufficient to effect the desired result.

If the urine reacts alkaline the alkali should be intermitted lest the formation of phosphates about existing stone occur. Lithia waters have been much in vogue, but lithia probably has no effect to dissolve the acid calculi present and probably does facilitate the formation of phosphatic calculi. As for the numerous mineral waters advised, one should assure oneself from reliable published analyses that they do contain appreciable quantities of alkalis.

If one wishes the effects of alkalis it is better to use them as such, citrate, acetate, or bicarbonate of potash, in doses of gr. xxx (2 gm.) every three hours until the reaction of the urine is reduced to near the neutral point.

As for piperazin and its compounds and other so-called uric acid solvents for which extravagant claims are made, I will express my total disbelief in their efficacy and dismiss them without further comment.

Renal Calculus

Of the various surgical procedures used for the relief of this condition mention only will be made here; for the indications and technic one must consult competent surgical authority.

The X-ray and ureteral catheterism are valued assistants in localizing the obstruction. The presence of a calculus should not precipitate a surgical procedure unless there are evidences at the time of its discovery of damage or, by virtue of its size, site, etc., a threat of damage to the kidney; for a stone in the pelvis may lie long quiescent or a stone in the pelvis or ureter may be passed without trouble or danger; but any calculus is rife with danger and the patient entertaining it should have adequate examination from time to time in order early to appreciate renal changes, infection and local injury. Moreover, the mere act of ureteral catheterism may so dislodge a stone from its point of impaction as to facilitate its passage.

Through the ureteral catheter local anesthetics such as cocain or eucain may lessen the spasm and effect a release, or lubricants may be introduced in the same way, such as sterile olive oil or almond oil.

Stripping the ureter through the vagina or rectum and dilatation of the ureter below the point of impaction have been attempted. Buerger has reported a fair measure of success with the latter method.

Among other measures that have met each with its modicum of success are cutting the meatus of the ureter to make more room, using a special ureteral forceps and even fulgurating.

Upon surgery we have to depend for relief in a large proportion of the cases. Ureterolithotomy, removal of stone from the ureter, and nephrolithotomy, removal of stone from the kidney, may be done when no infection or complication is present, with a minimum of danger.

Of the three operations on the kidney, pyelotomy, nephrolithotomy, and nephrectomy, the first, pyelotomy, or incision into the pelvis of the kidney, is indicated when the stone or stones are of small or medium size and confined to the pelvis of the kidney, and is the operation least to be dreaded and gives the best results.

When the stones are multiple, when branched into the calices and when infected nephrolithotomy may be done. When infection is present, however, the drainage sinus is loathe to heal, the infection often persists and thereby endangers the other kidney, so that nephrectomy becomes eventually imperative. When, then, infection is marked, with pyelonephritis, pyonephrosis, and perinephritis, a nephrectomy should be the operation of choice.

A nephrectomy, however, must be preceded by an assurance in the mind of the operator that the remaining kidney is capable of functioning, and entails on him the necessity of determining this fact to the best of his ability by the use of the functional tests, determination of urea—or total non-protein nitrogen in the blood, study of water, saline and nitrogenous output, while the intake is accurately known, and by the inspection of the remaining kidney if in doubt.

More serious to the patient and more trying to the therapeutist is the infection superadded to the nephrolithiasis, the treatment of which has been taken up under Pyelitis.

REFERENCES

Bevan. Journ. Am. Med. Ass., 665, Feb. 26, 1910.
Kahn. Arch. Int. Med., Jan., 1913.
Kahn and Rosenbloom. Journ. Am. Med. Ass., Dec. 31, 1912.
Matthes. Lehrbuch d. klin. Hydrotherapie, Jena, 34, 1903.
Rosenbloom. Journ. Am. Med. Ass., Oct. 9, 1913.

CHAPTER XXIX

TUBERCULOUS KIDNEY

Frank Sherman Meara and Alfred Townsend Osgood

If, in considering the relationship of kidney affection to tuberculosis, we accept the broad classification of Castaigne and divide the kidney lesions into those which show tuberculous lesions and those which, in a tuberculous subject, may be reasonably attributable to tuberculosis, such as amyloid kidney, parenchymatous or interstitial nephritis, it is only with the first class of cases that this article deals.

Even among the kidneys that show local tuberculosis a discrimination must be made therapeutically between those which share in a general miliary tuberculosis, are bilateral and not amenable to any therapeutic effort, and those which are sometimes called primary, better termed "chronic localized tuberculosis," which, at least in its incipiency, is unilateral in 90 per cent or more of the cases, and in that very fact enhances the chances of success in treatment immensely.

Of the three possible modes of infection, hematogenous, or descending, by the genito-urinary tract, or ascending, and by contiguity, it is the consensus of opinion to-day that in the vast majority of cases the route is by the blood-stream. The original site of infection may be obscure, as a gland, a bone lesion, or old pulmonary lesion, or more obvious.

The ureter and bladder may share in the process, while the involvement of the second kidney, so often due to infection through blood or bladder, from the first, is an important fact in determining surgical procedure.

TREATMENT

The treatment of tuberculosis of the kidney, like the treatment of tuberculosis of the lung, begins with the earliest possible appreciation of the incipiency of the lesion, so that accuracy of diagnosis becomes an essential part of the treatment, and too much attention cannot be given to the early manifestations of the trouble. The importance of this statement lies in the fact that the only curative treatment follows immediately upon the diagnosis, namely, extirpation of the affected kidney, and the earlier the more certain may one feel of complete eradication of the disease.

These earliest signs of tuberculosis are usually expressed as disturbances in the urine, either in its composition or its evacuation. So common are these urinary troubles in the experience of every practitioner that a pernicious habit has grown up of classifying such under the rubric of "cystitis" and treating their importance as trivial. So remote are these earlier manifestations from the disasters that eventually make plain the true state of affairs, and interrupted by so many periods of remission or amelioration, that their connection is often forgotten when the diagnosis is established. When one considers that these so-called cases of cystitis are the announcement of new growths, or stone in the bladder, hypertrophied prostate or of new growth, stone, or tuberculosis of the kidney, and that, even when the condition is true cystitis, pyelitis and pyelonephritis may ensue, one feels that an abnormal condition of the urine or its passage should always be dealt with seriously and investigated in the light of these possibilities.

The first symptom that may take the patient to the physician is pain, stiffness or soreness in the muscles of the lumbar region, and here again a besetting sin is the inclination to call all these pains lumbago, forgetting their relation to the kidney and other pelvic organs, as well as to disease of the spinal column and nerve roots. Again slight constitutional disturbances for which it is difficult to attribute a cause may be due to the involvement of the kidney in a tuberculous process.

The disturbances in the evacuation of urine that attract the patient's attention are the quantity, frequency, and discomfort attending it. *Polyuria,* not necessarily with pain, may be the earliest sign; indeed, in the great majority of cases it is the first symptom. During this early period of polyuria the urine shows no blood, no pus, and usually no tubercle bacilli. Frequency of micturition, especially at night, is fairly common. This is followed by a *period of pain.* The pain varies in degree and character, but mention is made by some authors of the rather characteristic occurrence of pain in the middle of the act of urination, relieved by its completion. Hematuria, microscopic or gross, occurs in this period.

Blood in the urine may first call attention to the trouble. Granted that, suspecting the kidney from any of these symptoms, the urine be examined and found abnormal, a search should be instituted for the cause.

The urine in these cases is acid and contains red and white cells. Pus cells in an acid urine excite the suspicion of one of two conditions, a colon bacillus infection or tuberculosis. Lawrason Brown says "that when a patient with tuberculosis develops albuminuria, renal tuberculosis should be thought of at once."

Cultures on the ordinary media will discover the bacillus coli, but not the tubercle bacillus, and the absence of growth increases suspicion and leads to a careful search for the tubercle bacillus, which, after repeated examination for its presence, is almost invariably found. *If the organism is not found the sediment should be injected into a guinea pig.* The red blood-cells are almost invariably present, intermittently in sufficient quantity to show macroscopically. Such hematuria is always highly suggestive of tuberculosis. Remissions are common, during which the urine clears up and the patient appears in the blush of health and the vigilance of the unwary physician is relaxed to the patient's ultimate undoing. A tenacious memory is a valuable item in the physician's armamentarium for sooner or later the symptoms will recur in more emphatic terms, for the process is almost certainly insidiously going forward to the eventual destruction of the kidney—and the patient. But the process is not confined to the kidney first involved during the symptomless periods, but the other kidney is implicated and the urinary tract in larger measure. A considerable portion of cases of male genital tuberculosis are secondary extensions of renal tuberculosis.

While the finding of the tubercle bacilli may be taken to establish the diagnosis of tuberculosis in the patient as certain and in the kidney as highly probable, still it is possible for the kidney to excrete the bacilli without itself becoming involved; moreover, tuberculosis of epididymis, spermatic cord, prostate or seminal vesicles may be the cause of the bacilluria; hence, a detailed examination of this tract is indicated. To localize the lesion a cystoscopy is imperative.

A cystoscopy determines the presence or absence of a bladder complication, while the examination of the ureteral orifices may indicate the side involved, which a ureteral catheterism makes more sure. The urine taken from these ureters is examined for the tubercle bacilli, blood, pus, etc. The function of either kidney must be determined by this method as well. The X-ray may add its assistance, and in doubtful cases the tuberculin test (hypodermatic) or inoculation of urine into guinea pigs will help. The best opinion urges operation at the earliest possible moment when one kidney alone is affected. The failure of satisfactory evidence of cure from medical measures alone, and the danger of increase of the condition with involvement of the other kidney and other structures urge the surgical procedure. The mortality of the procedure has steadily fallen to 5 per cent and less in some series, while the percentages of permanent cures have become encouraging. Neither the presence of tuberculosis in other organs, nor involvement of the bladder, forbids the operation; the bladder lesion heals the more rapidly after the removal of the kidney.

Involvement of the other kidney contra-indicates nephrectomy. The interpretation of the urine coming from the other ureter in determining the involvement of the second kidney demands at times nice judgment.

Brown, quoting Rovsing, says, "the absence of albumin, pus, or bacteria from the second kidney may be relied upon as a rule; if pus and bacteria are present removal of the first kidney is usually contra-indicated, while if albumin, but no pus or bacteria, is found, it is not a contra-indication, the albumin being due to toxic changes and usually disappearing rapidly after the removal of the diseased organ."

The functional tests are such as were discussed under Pyelitis.

As in the surgical treatment of pyelonephritis, the question of nephrectomy arises, and it seems to be that the pros and cons in the two conditions (see Pyelitis) are much the same, except that hesitation and delay in carrying out vaccination and other medical measures are less justifiable in tuberculosis of the kidney.

If an early diagnosis is not made or, being made, the operation is refused, the patient will sooner or later enter on a period of great distress. This is the period of "cystitis" due to the striking involvement of the bladder in the process. Robbed of rest and sleep by frequency of micturition, often every fifteen to thirty minutes night and day, worn with the pain that accompanies each voiding, burdened with the added toxins of secondary infections, especially in the kidney, and the febrile impulses accompanying such infections, the patients' health and strength are undermined and their state is most pitiable. Gross lesions are present in the pelvis of the kidney or its parenchyma by this time, and innumerable tubercle bacilli are present in the urine, and yet an army of these sufferers are ever treated for a "rebellious cystitis."

If the patient refuse the operation, or if the two kidneys be so involved as to make operation unjustifiable, and, indeed, after an operation, whether there be a residuum of tuberculous infiltration in the bladder or not, the patient is to be treated like a case of pulmonary or other form of tuberculosis. It is now generally believed that the so-called primary tuberculosis of the kidney is in reality always secondary to a process elsewhere in the body, as has been mentioned, and even when it is not evident it is to be assumed and the therapy to be determined by the assumption.

Such treatment consists of an abundance of fresh air, a sufficiency of good food, and a proper hygiene. To this may be added cautious vaccine treatment and the relief of symptoms of distress caused by the condition itself or by its complications. Fresh air has come to be known as the most important factor in the successful treatment of pulmonary tuberculosis, but it is not to be forgotten, as it sometimes is, that fresh air is equally important in the treatment of tuberculosis of other organs than the lung, and, indeed, that in so treating tuberculosis of other organs we are often, perhaps most often, treating an incipient or latent tuberculosis of the lungs. But I think we should go further than that and appreciate that a tuberculosis is not the only infection, acute or chronic, which is benefited by fresh air, as was insisted on in the chapter on pyelitis.

Patients, then, with renal tuberculosis should have an abundance of air both day and night, and the same devices for sleeping out of doors as are used in cases of pulmonary tuberculosis may be employed here, the porch, the veranda, the lean-to, the tent, the fire-escape, the roof or, failing these, the room with the maximum number of windows wide open and with all the precautions for protection used under such circumstances.

Sunlight, a pleasant environment, quiet, freedom from dust all add their increment to the cure of the patient.

The proper observance of hygienic measures is of importance. Baths should be taken daily not only for purposes of cleanliness, but also as a therapeutic measure. This latter result is obtained by the cold bath, which stimulates the nervous functions and improves the circulation. In the use of the cold bath, however, one must be guided by the condition of the patient and his reaction toward the procedure. These baths may be taken in the shape of a sponge, plunge, or shower.

The clothing of the patient should be determined by the practitioner, who, appreciating the necessity of avoiding chilling the surface of the body and at the same time preserving the tone of the skin, insists that underclothing shall be of wool, which readily absorbs moisture and, being a poor conductor of heat, allows the skin to cool slowly.

Next to wool silk is preferred for these qualities, while cotton and linen are to be avoided. Overloading with clothes keeps the skin constantly moist in its own secretions and has the relaxing effect of a tepid bath, so that in sudden exposure to the cold air the vessels do not contract and chilling ensues. Clothing indoors should be light and heavier garments added on going out.

A sufficiency of sleep and periods of rest must be afforded; exercise should be insisted upon, but always regulated to the individual patient and his condition and never carried to fatigue. The bowels should be regulated, first by habit and diet and, if necessary, by medication, but always by the mildest that proves effectual.

Diet.—The value of an abundant dietary in pulmonary tuberculosis no longer requires a militant advocacy; it is an accepted fact, but the value of the same dietary may not be equally appreciated in renal tuberculosis, where fear of overburdening the kidney is entertained. When one considers, however, that, of the three foodstuffs, only one makes demands on the kidney, the protein, and that this may be economized if there is any evidence of incapacity of the kidney in its elimination and, moreover, when one considers the readiness with which compensatory hypertrophy takes place in the kidney, he may dispel such fears and feed abundantly. He should remember that a man whose pursuits are light, such as intellectual work, requires about 3,000 calories of food and about 100 gm. of protein a day and just as much more in the condition under consideration as his appetite dictates and digestion handles and, further, it must be remembered that if our patient is febrile his demands are not less than those of a healthy man at rest, but more—on theoretical grounds his requirements should be as a minimum some 25 per cent more. In a damaged kidney, however, since economy in protein may be advantageously exercised, 60 to 80 gm. will probably be quite sufficient.

The importance of ingestion of water must not be overlooked, not only as necessary to proper sequence of metabolic events, but also for its specific effect on the kidney and urinary passages.

Tuberculin.—It is tempting to the practitioner to utilize this agent in combating the tubercular process in the kidney, but it must be emphasized that the results obtained so far in this condition do not justify the delay of surgical procedure when the latter is indicated. When, however, operation cannot or may not be performed tuberculin therapy finds a proper place in the treatment of the patient.

It cannot be reiterated with too much insistency that this procedure is not to be lightly undertaken; that it requires thorough knowledge of the rationale, accuracy in technic, and judgment in its application; that one undertaking the use of tuberculin for the first time should give the same conscientious study to the situation he would feel incumbent upon him in approaching his first appendectomy or hysterectomy, for as much harm can come to the patient from an ignorant or neglectful physician in the one case as from an ignorant and neglectful surgeon in the other.

If one cannot do this or has occasion to use tuberculin but rarely, it is better to elicit the aid of an expert in its administration, or not use it at all.

Symptomatic Treatment.—In inoperable cases our efforts are directed to rendering the patient as comfortable as possible. The hygiene and diet are considered as just set down and symptoms treated as they arise. Pain in the kidney region is rarely severe unless complicated with stone or abscess and then is treated with local applications and, if necessary, with drugs in the same manner as specified in the chapters on Nephrolithiasis and Pyelitis.

Still more rarely a hematuria sufficiently severe to demand attention may occur. Rest enforced by morphin hypodermically is by all odds the most important measure, which may be accompanied by the use of calcium salts, chlorid, gr. xv (1 gm.) by the mouth four times a day, or lactate, gr. xx (1.33 gm.) by hypodermic injection at the same intervals, horse serum in 10 c.c. doses, better yet, human serum in amounts of 10, 20, 50 c.c. or even whole blood into the subcutaneous tissue in 10 to 30 c.c. doses, gelatin foods by mouth to meet theoretical considerations dealing with enhanced coagulation. In very severe hemorrhage transfusion might be indicated.

Fever is best controlled by the use of cold water, as sponges, and open-air treatment; the chills by local application of heat at the time of their occurrence; sweats, which may be very annoying, by proper care of the body, avoiding too many bedclothes, open air, hot drinks at night, sponges on retiring to which vinegar may be added, ℨi to the pint or more, or alcohol sponges, 25 per cent, or camphoric acid in doses of gr. xx to gr. xxx (1.33 to 2 gm.), or, in most obstinate cases, by atropin in doses of gr. 1/150 to gr. 1/100 (0.00045 to 0.006 gm.).

Gastro-intestinal disturbances may be present, the anorexia taxing the skill of those who order and those who prepare the food, while constipation or diarrhea may require modifications in the diet, the use of laxatives on the one hand, or astringents on the other; but one should always use the mildest of these that are efficacious, cascara, aloin, compound licorice powder on the one hand, and bismuth on the other, before having recourse to drastic purges or opium.

Complications.—In the advanced cases the ingenuity and patience of the physician may be taxed to keep the patient even relatively comfortable in the presence of one or more possible complications. One of the most common is implication of the bladder.

Local applications of heat to relieve pain, the use of acids or alkalis to change the reaction of the urine, belladonna or hyoscyamus to diminish irritability, irrigations of the bladder, or even operation for drainage may be necessary, or the use of opium. Pyelitis and pyelonephritis, more rarely, failure of function of the kidney, with suppression and uremia, demand the efforts specified in the article on Pyelitis, or perinephritic or paranephritic abscess requires surgical procedure or may rupture into the peritoneum.

Prophylaxis.—Prophylaxis begins with the patient, who submits himself periodically to medical examination or at least on the occasion of slight ailment; then, on the conscientious examination of such patients and the discovery of incipient lesions of a tuberculous nature, the prompt removal of same, if possible, and detailed instruction as to mode of life and work.

Such precautions are doubly imperative in those who are subjected to a tuberculous environment or have a tuberculous history.

Both as a prologue and an epilogue to the article, the authors would insist, with the reiteration of Cato's "Cathargo delenda est," *suspect every case of abnormally frequent and every case of painful urination as being tuberculous,* at least to the extent of repeated, thorough, search of the urinary sediment for tubercle bacilli.

REFERENCES

Brown. Osler's Modern Medicine.

Brown, Lawrason. The Significance of Tubercle Bacilli in the Urine, Journ. Am. Med. Ass., March 13, 1915.

DISEASES OF THE BLADDER

CHAPTER XXX

CYSTITIS

EDWARD L. KEYES AND HOWARD S. JECK

PROPHYLAXIS

The prevention of infection of the bladder is by all odds the most important phase of its treatment, for the normal bladder may be spontaneously infected only by the direct extension of gonorrhea or tuberculosis from a neighboring organ (notably the prostate or the ureter) or by irruption of an adjoining abscess, while the diseased bladder (retention, stone, tumor) may be infected by any pyogenic organisms.

Prevention of Gonorrheal Cystitis.—This is accomplished by the rules, especially the hygienic rules laid down in reference to the treatment of acute urethral gonorrhea. It is to be noted that injections cause acute cystitis only when they are given brutally or to patients who already have a smoldering, unsuspected infection of the bladder neck.

Prevention of Infection by the Tubercle Bacillus.—Tuberculous cystitis threatens every patient with tuberculosis of the kidney or of the prostate and seminal vesicles. The proper treatment of the former is nephrectomy, but the latter must be treated by palliative measures.

Prevention of Spontaneous Pyogenic Infections.—The bacillus coli and the pyogenic cocci cannot attack the normal bladder, but if the bladder is congested or otherwise diseased, as is commonly the case in retention, stone, and tumor, it is peculiarly vulnerable to infection. The removal of the etiological factor will, of course, do away with the danger of infection, but such treatment is not always possible, notably in cases of large malignant tumors. Under such circumstances the common sources of spontaneous infection must be carefully watched. These are the prostate and bowel. There is ample evidence to show that constipation causes congestion or inflammatory lesions in the mucous membrane of the colon that permit absorption of the bacillus coli from the bowel. The bacillus passes through the kidney and reaches the bladder, there to cause cystitis if the bladder is not normal. Hence it is of the utmost importance to prevent constipation in such cases.

The second source of spontaneous infection, for instance, the prostate, may for years be the seat of a mild infection (usually due to the staphylococcus) without causing any symptoms whatever, yet forming an admirable focus of infection in case of any intercurrent diseases of the bladder itself. The thorough treatment of postgonorrheal prostatitis constitutes the prophylaxis here. As a further preventive of spontaneous infection it is sometimes useful to employ hexamethylenamin in doses of 20 to 30 gr. a day.

Prevention of Catheteral Infection.—We have already insisted upon the gentleness during catheterism, the asepsis before and the antisepsis after, which are required to prevent infection.

TREATMENT

Acute Cystitis.—Acute gonorrheal posterior urethritis usually extends over into the trigone and is the type of acute cystitis. The treatment of this condition is the treatment of acute gonorrheal cystitis. Acute cystitis from other bacteria usually arises in the course of a chronic inflammation of the bladder, excited by accentuation of the causes which are keeping up the chronic cystitis. Such are the familiar "fit of the stone," the acute cystitis following acute prostatic retention, etc.

The treatment of these conditions, of course, depends largely upon the existing cause. As a general rule, however, it may be stated that rest in bed, catheter irrigations of the bladder once a day with warm silver-nitrate solution (1: 10,000), or, if this is too strong, with potassium permanganate (1: 6,000), boracic acid, or argyrol, are necessary. Some alkaline diluent should be administered, and proper measures taken to relieve the cause of the acute attack.

Chronic Cystitis.—Since chronic cystitis is simply the manifestation excited by various bladder diseases (for example, retention, stone, tumor), the first point in the treatment is to decide whether the cause is to be cured or endured. In these surgical days most patients prefer the knife, and this ultimately relieves the inflammation of the bladder. But if the cystitis is to be endured, as in the case of inflammation due to prostatic hypertrophy under treatment by systematic catheterism, inflammation of the bladder must be controlled, whether by the use of one of the bladder washes mentioned above or by hexamethylenamin, or by both. Such treatment will in some cases keep the bladder absolutely clean; but if the bladder is sacculated, or gravely infected, a mild cystitis is likely to persist in spite of treatment.

Tuberculous Cystitis.—The treatment of tuberculosis of the bladder, which is never primary, ought to be chiefly directed toward the primary focus, be that in the internal sexual organs or in the kidney. But as in certain cases of genital tuberculosis or in bilateral renal tuberculosis, it is impossible to relieve the irritation caused by the primary focus, or if, after removal of the tuberculous kidney, ulcerations persist in the bladder, these are best treated by hygiene and the following local measures: (*a*) Instillation into the bladder every day of 5 to 10 drops of one of the following solutions, beginning at the minimum dose mentioned and gradually working up to stronger doses: Bichlorid of mercury 1:20,000 to 1:5,000; gomenol oil, 1 per cent to 20 per cent; thallin sulphate, 2 per cent to 12 per cent; iodoform in oil, 10 per cent; carbolic acid, 1 per cent to 5 per cent. (*b*) Cauterization. The ulcers are burned singly, either by means of the high frequency current (d'Arsonval or Oudin) or by acid nitrate of mercury. The latter is applied on a swab directly to the ulcer through a direct vision urethroscope. After locating the ulcer, the sheath of the instrument is used as a cofferdam by bringing its end directly in contact with the bladder mucosa. The ulcer is then swabbed with a dry applicator before using the acid nitrate of mercury.

As a last resort anastomosis of the ureter and skin has been resorted to to relieve the pain of tuberculous cystitis.

Hunner Ulcer.—This lesion, described by Hunner of Baltimore, when not too large may be cauterized by the high frequency current or by the acid nitrate of mercury by the method described above. If extensive, however, the ulcer may require excision. The latter operation frequently necessitates removal of a large portion of the bladder, for, since the ulcer is of the undermining type, the incision must be made wide of its surface margin.

Functional Neuroses of the Bladder

Three types of neuroses are worthy of consideration:

1. Enuresis.
2. Pain in the bladder or frequent and painful urination due to hysteria or neurasthenia.
3. The same symptoms due to reflex irritation.

Treatment of Enuresis.—Enuresis, or idiopathic incontinence of urine, is that condition in which the patient unconsciously empties his or her bladder without having any control of this act or any physical cause for the same. Enuresis commonly occurs only in children between the ages of three and twelve, and only at night. In severe cases it is both nocturnal and diurnal. Inasmuch as this condition is apparently a purely functional one, due to inadequate control on the part of the child, it is usually curable by the removal of any existing cause of irritation, such as adenoids, adherent foreskin, pin-worms, etc., plus proper training of the child. The elements of proper training are:

1. The child shall not drink any fluids after 5 o'clock in the evening.
2. It shall be instructed that the symptom is in some measure under its own control, so that it shall help as far as possible in the cure.
3. It shall be taken up at night often enough to prevent unconscious urination.

At first it may be necessary to awaken the child every half hour and, if so, this should be regularly done for a number of nights, and then the interval gradually lengthened.

Various drugs have been recommended in the treatment of this condition, among which doses of a tenth of a grain of the extract of belladonna, given t. i. d., and increased until the physiological effect is observed, bear the greatest reputation. Rhus toxicodendron, or aromatica, 20 drops of the fluid extract, three or four times a day, is also well spoken of. A few years ago a great deal was thought of Cathelin's treatment: injection of salt solution into the cauda equina through the terminal foramen of the sacrum, but this is little spoken of latterly. Thyroid extract has been used successfully.

Among other methods of treatment may be mentioned faradization of the urethral mucosa by means of an electrode in the membranous urethra, with the opposite pole over the pubes; mechanically shutting off the urethra by means of a clip which the patient has to remove in order to urinate, and making the patient lie with head low and pelvis high are worthy of mention.[1] But the backbone of the cure is intelligence and hygiene.

Treatment of Reflex Neuroses of the Bladder.—Neurosis of the bladder in the adult is usually due to some functional or organic disturbance of the sexual organs, and the only real difficulty in the treatment of these cases consists in the proper diagnosis of the cause of symptoms.

The causes may be divided into three types:

1. General neuroses.
2. Causes peculiar to the male.
3. Causes peculiar to the female.

Among general causes of bladder pains and irritations, fissure in ano indicanuria, colitis, intestinal adhesions, and phlebitis of the iliac veins are especially worthy of mention.

Among the causes peculiar to the male, prostatitis, vesiculitis, congestion or irritation of the verumontanum, and sexual neurasthenia may be mentioned.

---

[1] Faradization, one pole in the rectum or vagina, the other over the pubes, is very valuable, especially in hysterical subjects.—Editor.

Among causes peculiar to the female are pressure from fibroids or other tumors, exudates and abscesses, uterine misplacement, cystocele, and the pregnant uterus. Fissure in ano, while not a very common cause, is at times a very definite one, especially in women.

Reflex irritation of the bladder from inflammation or distention of the kidney pelvis should also be borne in mind.

Having identified the cause of the irritation, its removal, combined with hygiene and a little psychic influence to overcome the bad effect of habit, constitutes the treatment.

Paralysis of the Bladder.—Paralysis of the bladder must be distinguished from atony. Atony is weakness of the bladder wall resulting from urethral retention, of which the common causes are urethral stricture and prostatic hypertrophy, carcinoma, or bar. True paralysis of the bladder may follow diphtheria or may result from any spinal lesion, but the common cause is tabes. Incomplete retention in an otherwise healthy bladder, without urethral obstruction, is often a sign of beginning bladder paralysis, which is the first evidence of tabes.

Treatment in this early stage by arsphenamin will sometimes relieve the paralysis, but if retention of urine continues in spite of this, or if the case is seen only after tabes is well established, the paralysis must be treated by emptying the bladder systematically with a catheter as in the treatment of retention from inoperable prostatic hypertrophy.

CHAPTER XXXI

NEUROSES OF THE BLADDER

Charles Godwin Jennings

Paralysis of the Bladder.—Paralysis of the bladder is usually a result of organic disease of the central nervous system. Rarely it is a neurosis dependent upon reflected peripheral nervous irritation involving the lower segments of the sacral cord. Any disease or injury of the cord that causes paraplegia may be attended by vesical paralysis. Dependent upon the nature of the lesion, the paralysis may develop gradually or suddenly, and it may be partial or complete.

Paralysis is to be distinguished from atony of the bladder, a condition more common and met with as a physiological manifestation of the enfeeblement of advancing age, or in pathological states, as a result of the muscular debility of severe, exhausting acute and chronic diseases. It is true overdistention from atony will cause a temporary paralysis of the muscular wall of the bladder. In this condition, however, the muscular fibers alone, and not the nervous mechanism of micturition, are involved, and it should not be spoken of as a true paralysis.

The striking and important result of vesical paralysis from whatever cause is retention of urine. In all acute and chronic diseases of the nervous system, in comatose states, and in severe exhausting diseases such as typhoid fever and other infections the physician should be on the alert to detect a vesical paralysis in its incipiency and before extreme distention has injured the bladder wall and rendered it liable to infection.

Symptoms of retention call for immediate examination of the lower abdomen to detect distention. If the bladder is palpable, catheterism is the remedy for immediate relief. The operation should be done under aseptic precautions. So long as the paralysis continues the catheter should be passed every eight or twelve hours, and, in conditions attended by polyuria, such as diabetes, more frequently. The condition is serious, for, sooner or later, the bladder becomes infected and the too familiar train of distressing symptoms follows.

Medical treatment of the condition is of minor importance, although urotropin in full doses will materially aid in preventing or delaying infection. The remedy should be administered as soon as catheterism is necessary. Permanent relief of the paralysis will be dependent upon the course of the primary disease causing the symptom. Remedies directed to the bladder alone are of doubtful utility. Electricity, ergot, and strychnin have been suggested.

ENURESIS

Enuresis occurs in childhood as a *neurosis,* the result of hypersensitiveness of the centers of micturition in the cord, with deficient control by the cerebral cortical centers; or as a *symptom* of various pathological conditions of the urinary and other organs.

The nervous mechanism of micturition consists of: (1) The spinal center in the gray substance of the third, fourth, and fifth sacral segments; sympathetic centers, which are cell complexes inserted in the hypogastric plexus; and cortical and subcortical cerebral centers, which communicate with the spinal centers through fibers in the anterolateral columns of the cord. (2) The afferent limb of the reflex arc, consisting of sensory fibers distributed to the bladder wall, which enter the cord with the posterior roots of the second, third, and fourth sacral segments. (3) The efferent limb of the reflex arc, carrying motor fibers to the sphincter vesicæ and detrusor urinæ, which reach the bladder by the pudendal nerve and the inferior hypogastric plexus.

In infancy urination is a reflex act. The urine is retained in the bladder by the tone of the elastic tissues surrounding the neck of the bladder and the urethra, and by the tonic contraction of the sphincter vesicæ. When intravesical pressure reaches a certain point afferent impulses ascend to the micturition center in the cord. From this center efferent inhibitory impulses are sent to the sphincter causing the muscle to relax, and motor impulses to the muscular wall of the bladder, which then contracts and the bladder empties. At about the end of the second year the spinal reflex mechanism comes under the control of the cerebral centers and, within certain limits, the urine is retained, or the bladder emptied, at will.

The age at which the cerebral control is established depends upon the inherited nervous stability of the child and to some extent upon the care with which he has been educated. After the third year inability to control the discharge of the reflex spinal mechanism of micturition under ordinary conditions constitutes enuresis. During sleep cerebral control is lessened, and at this time causes that excite reflex discharge are most active. Nocturnal enuresis is the more common form of the disorder. Constant enuresis, diurnal, as well as nocturnal, is frequent. This form may be due to mental enfeeblement or to some serious disease of the central nervous system. Spina bifida occulta is an occasional cause and is incurable. Epilepsy as a cause of nocturnal incontinence should be kept in mind. Incontinence appearing first after the age of five years and appearing only at irregular times may be of this character. The treatment of cases dependent upon these and other serious diseases of the nervous system belongs to a different chapter.

When it is impossible to ascribe to enuresis an adequate cause, the condition is considered as a neurosis—*essential or idiopathic enuresis.* These cases may be considered as due to the persistence of the infantile mechanism of micturition. With a searching investigation into the clinical history and a complete physical examination, the number of idiopathic cases will diminish, and the physician should not be content with such a diagnosis until all methods of investigation have been exhausted. Still it must not be forgotten that, while in some cases the irritating pathological condition is manifest and would excite an enuresis in a normal child, in most cases the exciting cause is trivial, and the important factor in the condition is the underlying nervous instability of the child. This nervous instability may be inherited or acquired. The inherited influence is shown by the fact that the parents of these children sometimes give a history of enuresis in childhood and that other children of the family also are afflicted. Other functional nervous diseases, such as chorea, habit spasm, and night terrors, may be present. An acquired nervous instability may be the result of any debilitating acute or chronic disease, or the nervous worry and strain that come with school life. It is quite often noted that an enuresis improves or disappears during the holiday season only to relapse with the beginning of the school period.

Treatment.—Treatment of enuresis should be preceded by a careful, systematic investigation into all the possible conditions that may cause it. Treatment directed to this symptom alone, without regard to its cause, usually results in failure. After more or less persistence it is given up, the condition looked upon as incurable by the parents, and the child is left to drift along with a distressing complaint until it is relieved by an accidental cure of the exciting cause or by the complete development of the higher centers of the reflex control that comes with puberty.

The various diseases that may be the exciting cause of enuresis, aside from the diseases of the nervous system mentioned above, may be considered under the following heads:

Hygienic: bad training, excessive intake of fluid, faulty diet.

Urinary changes: hyperacidity, alkalinity, bacteriuria.

Diseases of the urinary and pelvic organs: atony of the sphincter, irritability of the bladder, phymosis, adherent prepuce, adherent clitoris, urethritis, cystitis, pyelitis, vulvovaginitis, vesical calculus, intestinal parasites, rectal polyp, anal fissure, chronic interstitial nephritis.

Diseases of remote organs: adenoids and hypertrophied tonsils, thyroid insufficiency, chronic gastric and intestinal indigestion, chronic constipation, diabetes mellitus and diabetes insipidus.

Hygienic.—A child must be taught to control its bladder and to make known its desire to empty it. Carelessness in this regard may result in a prolongation of the habits of infancy. Properly trained, the child should have the mechanism of micturition under cerebral control by the end of the second year.

A faulty diet will be found to be the determining factor in many cases in which the enuresis first appears after the age of four or five years. Dietetic errors cause incontinence in predisposed children by inciting chronic intestinal indigestion with resultant absorption of the products of excessive intestinal putrefaction and fermentation. Constipation, flatulence, an irritating urine, together with the exaggerated irritability of the reflex center, resulting from impaired nutrition, determine the incontinence. A diet in which carbohydrates, particularly sugar and sweets, are in excess is the most frequently observed fault. The writer has seen many cases promptly cured by a reduction in the starch and sugar content of the diet. Elimination of the sugar alone is sometimes successful. Excess of meat is more rarely a cause. In every case of enuresis the diet should be carefully prescribed. There should be such an adjustment of proteins, fats, and carbohydrates as to bring about the best nutrition of the child with the least tax upon the digestion. Excess of fluids should be avoided, although it is useless to attempt to control enuresis by restricting the fluids to a quantity below the physiological requirement. The food should be bland, and all stimulating articles, such as tea, coffee, spiced and seasoned foods, and meat juices, should be forbidden.

Urinary Changes.—Vesical irritability may be caused by polyuria, hyperacidity, or alkalinity of the urine, and bacteriuria. A careful analysis of the urine, chemical and microscopical, should be a routine procedure in the examination of every child with enuresis. The necessity for this cannot be emphasized too strongly. The routine tests for albumin and sugar are not enough. A hyperacidity, a few cells or an excess of epithelium indicating a mild vesical catarrh, or infection with the colon bacillus may be the exciting cause of an enuresis and escape detection unless carefully sought for.

Polyuria may result from too much fluid in the diet, as noted above. Diabetes mellitus will be revealed by the urinary analysis. Chronic interstitial nephritis, although a rare cause of nocturnal polyuria with incontinence, should always be kept in mind.

Hyperacidity of the urine, with heavy deposit of urates, uric acid, or oxalate of calcium, is associated with many cases of enuresis and may be an important factor in its production. This condition may attend chronic derangements of digestion, or an excess of meat protein in the diet. Such cases demand a careful diet prescription with an alkali: acetate or citrate of potassium with tincture of hyoscyamus.

Alkalinity of the urine occasionally is a cause of vesical irritation. It occurs with polyuria and a urine of low specific gravity, which deposits triple phosphates and may contain a trace of albumin. Guthrie states that a characteristic of this condition is the quick repetition of the discharge of a large quantity of urine after the child has been awakened to empty the bladder. This condition may be the urinary manifestation of an excess of starch or sugar in the diet.

Microscopic examination of the stained centrifuged sediment of the urine shows that infection of the urinary tract by the colon bacillus is an occasional cause of enuresis. The urine, when passed, is clear, highly acid, has an offensive odor, and stains the clothing. On standing, it quickly becomes turbid, and its reaction changes. There may be a trace of albumin. This condition may escape detection because of imperfect examination and the failure to appreciate that infection may be present without pus in the urine.

The treatment of colon bacilluria is the administration of full doses of alkali or of urotropin. Five to 10 gr. each of acetate and citrate of potassium, well diluted with water, may be given before each meal and at bedtime. Larger doses may be given in obstinate cases. Hexamethylenamin, or urtropin, is the most useful urinary disinfectant. The drug is inert if the urine is alkaline. If the urine is acid, urotropin breaks down to form formaldehyd and has a strong antiseptic action. Acid sodium phosphate increases the acidity of normal urine and renders an alkaline urine acid. Urotropin, therefore, should be administered with acid sodium phosphate. The minimum adult dose of urotropin necessary to disinfect the urine is 15 gr. three times a day. The minimum adult dose of sodium acid phosphate necessary to acidify the urine is 30 gr. three times a day. Individual unpleasant reactions to urotropin are often observed. In doses sufficient to disinfect the urine it may increase vesical irritability, and occasionally it has no antiseptic action. It is often useful to alternate the administration of alkalis and urotropin every week.[1] Tincture of hyoscyamus or belladonna in elixir of buchu may be given at night to lessen the irritability of the bladder. At the same time the bowels should be kept free and the colon flushed two or three times a week to prevent reinfection. While rarely necessary in a simple bacilluria, in obstinate cases the administration of a colon vaccine after the method spoken of under Cystitis and Pyelitis may be advisable.

Diseases of the Urinary and Pelvic Organs.—Atony of the sphincter is a condition not easy to demonstrate, but may exist in feeble children, convalescent from acute illnesses, or suffering from chronic nutritional diseases. In this condition the urine is frequently voided in small quantity both day and night. These cases may be relieved by the general measures recommended below, directed to improving the tone of the muscular and nervous systems, and by local treatment to the sphincter itself. The faradic or galvanic current may be of use. The application may be made with one pole attached to a large flat electrode placed over the lumbar or suprapubic region and the other attached to a small electrode applied to the perineum. Some cases do better with a vaginal, urethral, or rectal electrode. Care must be used to begin with the galvanic or a very mild faradic current, and gradually accustom the child to it. The discomfort and fright produced by an injudicious application at first may make further treatment impossible. In some cases massage of the neck of the bladder through the rectum may have a marked tonic effect on the sphincter, and at the same time relieve a catarrh of the deep urethra and prostate.

---

[1] The forcing of liquids is an important part of the treatment.—Editor.

Excessive irritability of the bladder may be of nervous origin, but usually it is a symptom of infection along the urinary tract. The irritability may persist after an infection of the urinary tract or some remote cause of reflex irritation has disappeared. Many cases of enuresis in which it is impossible to find an adequate cause may be explained in this way. The cause has been removed but the irritability that resulted from it remains and the bladder is excited to contract by the pressure in it of a small quantity of normal urine.

The treatment of an irritable bladder after the exciting cause of enuresis has been removed consists in the administration of vesical sedatives and the education of the bladder to endure greater distention. It is often advantageous to keep the child in bed for a few days and instruct it to void at gradually increasing intervals. In cases that have persisted for some time and resist other measures the injection into the bladder of increasing amounts of a sterile salt or boric acid solution will be followed by good results. The injection may be made through a small soft catheter, or, in older boys, by hydrostatic pressure without the introduction of the catheter.

Urethritis, cystitis, and pyelitis are revealed by urinary examination and by pain and other symptoms referable to different portions of the urinary tract. Urethritis is an uncommon cause, although it may be present in older boys with long-continued enuresis, or as a result of bladder infection by the colon bacillus. Very rarely there is a gonococcus infection. Deep urethritis in older boys may require the passage of a steel sound or the injection of argyrol (10 per cent) or silver nitrate (1 per cent) into the deep urethra. The well-known technic of modern genito-urinary surgery should be used. These measures sometimes promptly cure an enuresis.

In addition to the administration of urotropin and the salts of potassium, colon infection of the bladder and pelvis of the kidney may be treated by a colon vaccine. A stock vaccine, or, when possible, an autogenous vaccine, may be given. Ten millions is a suitable dose for a child of five years. This may be repeated at intervals of four days for several doses, and the number increased to fifteen millions. The results of vaccine treatment of colon infection of the urinary tract are often brilliant.

Certain obstinate cases may be caused by a vesical calculus. An elongated and adherent prepuce and phimosis in boys, or an adherent clitoris in girls, is often present in enuresis, and is the local irritant that maintains the hyperexcitability of the micturition reflex. They should always be sought for, and, if present, relieved. Circumcision should be performed in cases with redundant prepuce and phimosis. Harmonic has reported 187 cases of enuresis treated by circumcision, of which 130 were cured, and many other writers speak with equal confidence of its beneficial effect.

According to the writer's experience, the results of operation alone will often be disappointing, and too much should not be promised from it. Phimosis should be looked upon as one of the factors, sometimes the most important, of incontinence. Even when circumcision is performed, treatment for the neurosis will usually be necessary to complete the cure. The pathological irritability of the nervous mechanism of urination that has been set up by the prolonged local irritation will require careful treatment on the general lines laid down elsewhere. Without circumcision, however, all efforts to overcome the condition may be unavailing.

Persistent balanitis or vulvovaginitis may be the exciting cause of enuresis, and certain cases may be traced to irritation of the lower intestinal tract. Parasites, particularly the oxyuris vermicularis, should be sought for and properly treated. A rectal polyp or an anal fissure is a rare exciting cause. Constipation is a common complication, and may be an important etiological factor. The influence of a loaded rectum may be readily noted in mild cases. Here it often will be noted that the incontinence relapses or is aggravated by a period of sluggish bowels. Relief of constipation should be a part of the routine treatment of every case. When the relation between the two symptoms is marked the thorough emptying of the rectum before bedtime may alone cure enuresis. In the absence of mental deficiency, cases showing diurnal as well as a nocturnal incontinence are usually due to some severe irritation along the urinary tract or the contiguous structures. Dysuria also points strongly to a local cause.

Diseases of Remote Organs.—Children with adenoids and hypertrophied tonsils are more liable than others to suffer from incontinence. Fisher found enuresis in 106, or 14.8 per cent, of 716 cases operated upon, and several other writers have reported about the same average percentage. Occasionally the enuresis is due solely to this cause, and operation promptly will be followed by relief. More often this is only one of the factors or an associated condition having no etiological significance. As in circumcision, the physician should be careful not to promise a cure by operation alone. Too frequently removal of the adenoids has no influence on the course of the enuresis. Such a result is discouraging to parents who have been assured of benefit and may lead to the abandonment of other essential therapeutic measures.

When adenoids cause respiratory obstruction they should be removed. The partial asphyxia produced by them during sleep stimulates the vasomotor center in the medulla, and excites a polyuria, an important factor in many cases of incontinence.

One of the most interesting of recent observations is the curative effect of desiccated thyroid in certain cases of enuresis. Hertoghe, of Antwerp, first saw beneficial results from its use. Leonard Williams, of London, reported 25 cases, only 1 of which failed to be benefited. The treatment was suggested to him by the fact that a boy operated for tonsils and adenoids for the cure of enuresis was made worse. Reasoning that the removal of the adenoids and tonsils had deprived the boy of a necessary internal secretion, thyroid was administered to make up for the loss. The boy was promptly cured.

Ruhräh treated a small series of cases and noted remarkable results in children presenting signs that might be attributed to thyroid insufficiency.

The writer has not had an opportunity to try out this remedy, except in two cases, both girls from four to five years old, with intractable incontinence from infancy, but without symptoms to indicate thyroid insufficiency. They were not influenced by careful thyroid medication extending over a period of three weeks.

Various disturbances of function of different organs in the adult resulting from thyroid insufficiency are so often markedly benefited by thyroid feeding that the experience of Williams should be given serious attention. As described by Williams, children with thyroid insufficiency are undersized and under weight. Their vitality is low and they continually show a temperature one or two degrees below the normal. They complain of cold, even in summer, and often have cold, blanched fingers. Many of them have adenoids and show a high-arched palate. Mental sluggishness is not characteristic. Such cases and others of obscure etiology in which well-directed therapeutic efforts have failed may be experimentally treated with thyroid. To a child of six years ½ gr. of desiccated thyroid may be given morning and night, and the dose increased gradually to 1 gr. three times a day. It is better to begin with a small dose, as overdosage has caused in some cases an aggravation of the symptoms. Favorable results should come within two or three weeks, and it is unnecessary to continue the remedy beyond this time if no effect is produced. According to Williams, cases favorably influenced by thyroid often show quite a remarkable improvement in nutrition and a large gain in weight.

Chronic gastric or intestinal indigestion is a predisposing rather than an exciting cause of enuresis, although it may act in both ways. The impaired nutrition, deranged metabolism, and anemia that result from indigestion render the reflex centers highly sensitive to irritation and make possible an enuresis from an insignificant local irritation; or the pathological changes in the alimentary tract may in themselves act as the reflex irritant that excites the involuntary emptying of the bladder.

General Treatment.—All cases of enuresis will require systematic therapeutics of a general character in addition to the measures instituted directly for the relief of the specific etiological factor, if one can be found. The condition is so complex, so often the result of a combination of causes, that it is best to take advantage of all means to diminish the hypersensitiveness of the mechanism of micturition.

Certain routine instructions should be given for all cases. While punishment in cases that have continued for any length of time is universally admitted to be more harmful than otherwise, certain careless and lazy children may well receive judicious discipline. Moral treatment, an appeal to its sense of delicacy, and some system of rewards will materially assist the child's cerebral control. A cold spinal douche with a brisk rub may be given on retiring and the rectum unloaded by a warm oil enema. The child should be awakened late in the evening to empty the bladder. The room in which the child sleeps should not be too cold, and he should be plentifully supplied with bed clothing. Elevation of the foot of the bed may be of use, but is often impracticable. Some mechanical device to prevent the child from lying on the back has occasionally been successful. An electrical device composed of two metal disks separated by absorbent cotton or some fabric connected with a battery and bell has been found successful by Genouville. Saturation of the fabric connecting the disks with urine completes the circuit and the bell rings.

Devices of this kind probably act by suggestion or by putting the cerebral centers on the alert for the impulses from a full bladder.

Every effort should be made to give these children quiet, invigorating surroundings. A life in the country is to be selected whenever possible. The nervous tension of school life should be relieved or ameliorated, and all exciting occupations and amusements forbidden. A holiday at the seashore, with freedom from the restraints of conventional city life, is often sufficient to effect a cure. Restriction of fluids in the latter part of the day has a good effect. No fluid except what is contained in the food should be allowed after four o'clock in the afternoon, and broths, milk, or other liquid foods should be limited to the lowest possible quantity. Careful inquiry should be made into the dietetic habits of the child, and every article that may be a source of irritation to the urinary tract excluded. As above noted, sweets and highly seasoned food are often the unsuspected offenders. A simple, plain, but well-balanced and digestible diet should be prescribed. Gastric and intestinal indigestion should receive the most approved treatment.

Many children with enuresis are robust, well nourished, and otherwise well. Here it is unnecessary to give tonics or otherwise to burden the parents with useless restrictions. Local irritation of some nature is the cause of the trouble and should be diligently sought and remedied.

An undernourished and anemic child should be built up by iron, arsenic, and cod-liver oil, with rigid attention to its food and digestion. Enuresis and night terrors often go together, and reveal the nervous origin of the trouble. Bromid of sodium, 5 to 10 gr., before supper and at bedtime may act beneficially.

Drugs.—Of drugs that directly influence enuresis, belladonna is the most frequently successful. The drug apparently owes its beneficial influence to its sedative effect on the muscular wall of the bladder. It may be given in the form of tincture of belladonna or atropin sulphate. Tincture of belladonna may be given to a child of five years of age in a dose of 5 minims three times a day, and increasing the dose by 1 minim every second day until flushing of the face from twenty to thirty minutes after administration shows that the full physiological effect has been obtained. It may then be reduced 1 minim a day until the flushing ceases, and this dose continued for several days. If no effect is produced after the drug is pushed to the limit, it is useless to continue it. It is unwise to keep a child for weeks in a state of mild belladonna intoxication. Atropin sulphate may advantageously take the place of belladonna. A common way to prescribe it is 1 gr. to 2 ounces of water. One minim of this solution will represent about 1/1,000 gr. of the drug. Two minims may be given as the initial dose to a child of five years, increasing 1 minim every second day, as directed for the administration of tincture of belladonna. The writer prefers to give the remedy in a vehicle, such as elixir of buchu or santal, with 5 to 10 gr. of acetate of potassium, which acts to allay irritability of the mucous membrane of the bladder. Tincture of hyoscyamus may replace the belladonna when there is infection along the urinary tract. It may be given in a dose double that of belladonna. Tincture of nux vomica or strychnin sulphate is indicated in all cases in which an atony of the sphincter is a factor in the enuresis. Strychnin acts also as a tonic to the hypersensitive centers in the cord. Freyberger, in cases that have resisted belladonna, has found good results from fluid extract of rhus aromatica in doses from 5 to 10 minims to a child of five years. He believes the drug acts as a powerful tonic to the bladder, similarly to nux vomica. In cases of nocturnal incontinence the doses of belladonna may be given in the latter part of the day so that the child will be under its full influence during the sleeping hours and thus escape some of its unpleasant effects.

Ergot has been given as a vesical tonic in cases of atony of the sphincter. In the writer's experience it has not proved of value when belladonna and strychnin have failed.

CHAPTER XXXII

THE TREATMENT OF TUMORS OF THE BLADDER

Howard S. Jeck

Before considering the treatment of bladder tumors, it is well to classify them briefly and to review their symptomatology.

For practical purposes the classification of Geraghty[1] is very satisfactory:

Tumors of Epithelial Origin

Papilloma
Superficial non-infiltrating papillary carcinoma
Localized circumscribed infiltrating carcinoma
Squamous-celled carcinoma
Basal-celled carcinoma
Adenocarcinoma
Cysts

The first four tumors are usually papillary in origin and comprise about 90 per cent of all bladder tumors.

Tumors of Connective Tissue Origin

Sarcoma
Myxoma
Fibromyxoma

All of the latter group are very rare.

SYMPTOMS

In the order of their importance, the chief symptoms caused by tumors of the bladder are hemorrhage, dysuria, pain, retention and cystitis.

Hemorrhage.—In the vast majority of cases the first and certainly the most prominent symptom of bladder tumors is hematuria. It may vary from a slight intermittent type to a hemorrhage so profuse that blood transfusion may have to be done to save the patient's life. But the characteristic type is sudden, profuse, painless hemorrhage which comes without warning and frequently stops as suddenly as it began.

---

[1] From a paper on Bladder Tumors read at the N. Y. Urological Society Meeting, March 26, 1924; also personal communication.

The amount of blood lost is not indicative of the size or character of the growth. Very small non-malignant papillomata may bleed so profusely that there is soon an acute retention due to clots, while a malignant growth filling half of the bladder sometimes gives rise to so little blood that the urine is merely tinged.

Dysuria.—Painful urination, except when due to an attempt to pass blood clots, is usually a much later symptom than hematuria. The same factors which cause pain on urinating are also responsible for increased frequency of urination which may vary from once every two or three hours to once every five minutes. Thus the factor of frequency is usually combined with the element of pain in the stereotyped expression "frequent and painful urination."

Pain.—Aside from the pain experienced during the act of voiding, there is also present in some instances more or less constant pain. This is felt in the suprapubic region or it may be of the radiating type and noticed only in the thighs and legs. It is about the last of all the symptoms to appear and is often absent entirely unless the growth is very large or has infiltrated deeply into the bladder muscle or unless metastasis has taken place.

Retention.—A large clot attempting to engage in the urethra or the tumor itself, when situated near the urethral orifice, may suddenly arrest the flow of urine.

The retention may be complete or partial; cystitis develops and the behavior of the patient in many instances may resemble that of one suffering from prostatic hypertrophy.

Cystitis.—While this condition commonly develops late, it rarely is one of the first symptoms. "The tumor is a point of least resistance. Instruments introduced into the bladder often bring infection with them and thus, in one way or another, at one time or another, cystitis occurs." (Keyes Urology 1923.)

The urine becomes ammoniacal and incrustations of the growth take place. As a result the patient suffers from frequent and painful urination of the most harassing type, passing for the most part only small quantities of foul, stinking urine full of pus.

Constitutional and Renal Symptoms.—If the growth be malignant, cachexia, as in malignancy elsewhere, may develop. If the hemorrhage from the tumor is profuse or even slight, but of long duration, the patient becomes pale and anemic.

When there is cystitis, especially in the presence of urinary retention the infection is prone to spread to the kidney.

Also the growth in the bladder may involve the ureteral orifice and cause either partial or, in time, complete blocking of the ureter. Hence kidney involvement, ranging from simple pyelitis and hydronephrosis to acute suppurative nephritis and pyonephrosis, may result.

NON-OPERATIVE CONTROL OF HEMORRHAGE FROM BLADDER TUMORS

The non-operative control of hemorrhage from a bladder tumor consists in emptying the bladder of clots in the hope (often realized) that the bleeding may then cease. Such treatment is but palliative and is intended only to tide the patient over until radical measures may be instituted.

When it is definitely known by the use of the cystoscope that the hemorrhage arises in the bladder and does not come from the upper urinary tract, the following method may be successful.

Under aseptic conditions, pass a large silk catheter (20F to 22F) with a large eye or preferably two or three large eyes. If the clots are small and there is sufficient urine in the bladder, they may pass immediately through the catheter. Should no clots pass through the catheter, it may mean that either the catheter has become plugged or that the clots are too large to become engaged in the instrument. Therefore suction may be made by means of a Janet syringe or one of that type. If this maneuver proves unsuccessful, then the injection of small amounts of any sterile or suitable antiseptic solution (boric acid, 1 : 5,000 acriflavine, or 1 : 5,000 potassium permanganate) with subsequent suction after each injection may accomplish the end desired.

When the catheter fails, an excellent combination for the removal of clots from the bladder *per urethram* is an evacuating tube and an evacuating bottle such as are used in the operation of litholapaxy. After passing the tube, it is connected with the bottle (the Bigelow aspirator, preferably) which has been filled with the irrigating solution. With the eye of the tube just within the bladder neck, the rubber bulb is alternately sharply squeezed and released. When the glass receptacle underneath the aspirator becomes filled, the entire instrument should be emptied and refilled with fresh solution. If a litholapaxy evacuation tube is not at hand, a satisfactory substitute is the sheath of the Brown-Buerger cystoscope. After emptying the bladder of clots, the injection of a dram or two of 1 : 10,000 adrenalin solution might prove helpful in further controlling the bleeding. Coagulen, another of the commercial hemostatics, might be tried. In certain instances of slight hemorrhage, I have thought acriflavine in 1 : 5,000 solution acted as a hemostatic and for that reason prefer it as the irrigating medium.

METHODS OF TREATMENT

I. **Fulguration.**
II. **Operation.**
III. **Radiation.**
IV. **A Combination of any of the above methods.**

The choice of the procedure depends on the type, size, location, stage of advancement of the growth and the condition of the patient.

FULGURATION

This method was described by Dr. Edwin Beer, of New York, in 1910, and is now quite universally employed. It is known also by the terms high frequency, electrocoagulation, desiccation and sparking, which are in a measure descriptive of what actually takes place.

Two forms of current are used, the Oudin or monopolar and the d'Arsonval or bipolar. The latter burns more deeply and is more destructive. For most of our work, we prefer it to the former.

"*Fulguration should never be performed under ether anesthesia.* In several unreported instances a spark flying from the patient's lips to the metal mouthpiece of the ether bag has ignited the ether in the patient's lungs, resulting in explosion of the chest and immediate death." (Keyes-Urology 1923.)

Novocain used locally will suffice as an anesthetic in most instances, as the pain which attends fulguration is chiefly the pain of cystoscopy.

About 2 c.c. of a 10 per cent solution of novocain is injected into the posterior urethra by means of an instillator. Employing a small rubber-bulb, blunt-pointed urethral syringe, 7 or 8 c.c. of 10 per cent novocain solution is now injected into the anterior urethra. The urethra is then compressed just back of the meatus by means of the thumb and forefinger and held tightly for at least five minutes.

If the additional instrumentation entailed by passing the instillator is thought to be unwise (there are some patients who will not tolerate the passage of more than one instrument at a sitting), then the anterior urethra only may be injected. In this event an attempt should be made to milk some of the solution back of the cut-off muscle while still compressing the meatus.

Either spinal, sacral or parasacral anesthesia may be successfully employed in those cases that are too sensitive for local anesthesia.

Technic of Fulguration.—The patient is placed in the lithotomy position as if for cystoscopic examination. The usual aseptic precautions are employed. If the bipolar current is used, the metal plate, which is connected to one of the poles of the fulguration machine, is placed next to the skin underneath the patient's buttocks. The other pole is connected to the electrode.

The cystoscope is passed, the obturator withdrawn and the telescope carrying the electrode is inserted. After locating the tumor, the end of the electrode is placed firmly against it and the current turned on by means of the foot switch. (See Illustration III.) Different portions of the tumor are fulgurated from a few seconds to half a minute, the length of time depending on the rapidity of the burning and whether or not the patient feels it. The nearer the point of the electrode approaches normal bladder tissue, the more painful the procedure becomes. When tumor tissue alone is being burned, the patient rarely is conscious of any pain.

The electrode is applied to different areas of the tumor and the different points of fulguration should be so spaced that the areas of necrosis which follow will overlap each other, though this is dependent to some extent on the accessibility of the growth.

If more than one treatment is necessary, it may be repeated in about a week, provided the patient's condition will permit. Should symptoms of cystitis, such as frequent and painful micturition, follow fulguration, then it is obviously wiser to wait until the bladder has quieted down before fulgurating again.

Active bleeding from a growth may obscure the field of operation and render the treatment very difficult, if not impossible. When this occurs, the bladder should be washed quickly and repeatedly with small amounts of solution in an effort to locate the bleeding point. The latter found, the electrode is applied gently to this area and the surface burned lightly. This procedure will frequently control the hemorrhage, whereupon the balance of the operation may be completed without interruption from this source.

An almost immediate blanching of the tissues where the electrode is applied shows that fulguration is actually taking place. This is accompanied also by bubbles of gas which emanate rapidly from the point of contact.

Troublesome hemorrhage may be caused by too strong a current. This may be avoided by shortening the spark gap.

The spherical electrodes of McCarthy are said to give more satisfactory results than the pointed electrodes of Bugbee.

Should short-circuiting occur, use a dry-cell battery instead of the street current for lighting the cystoscope.

OPERATION

The term "operation," as used to-day in connection with the treatment of bladder tumors, may imply:

a. The exposure and opening of the bladder as a palliative measure as in the control of hemorrhage and relief of retention due to blood clots (suprapubic cystotomy).

b. The surgical exposure of the growth and its excision (by knife or actual cautery) or its destruction by the actual cautery.

c. The surgical exposure of the growth for the implantation of radium (the element itself or radium emanation).

d. Complete removal of the bladder (cystectomy) and transplantation of the ureters.

e. The establishment of a suprapubic fistula (cystostomy).

Suprapubic Cystotomy—*For Control of Hemorrhage and the Relief of Retention.*—In cases where catheterism and irrigation fail to relieve the hemorrhage, or in acute retention due to blood-clots which cannot be washed or aspirated from the bladder through a urethral instrument, operation should be resorted to.

If the loss of blood has been so great that the patient has become noticeably anemic, a red cell-count and hemoglobin estimation should be made. Then, if necessary, a blood transfusion should be done. It is oftentimes advantageous to perform the transfusion a few hours before the operation rather than after the operation, as the patient is thereby fortified to stand the operative shock.

Except in highly nervous individuals, the opening of the bladder and removal of the clots may be done under local anesthesia; but where a general anesthetic is required, gas and oxygen is the anesthetic of choice.

Technic Under Local Anesthesia.—If the patient is *in extremis,* no time should be lost in carrying out the usual procedure of irrigating the bladder and then refilling it with a sterile solution or air. In such cases it is usually found distended sufficiently to render the exposure of the bladder relatively easy.

The skin in the mid line of the abdomen is infiltrated with a 1 per cent novocain solution, beginning at the upper margin of the pubic bone and extending upward for a distance of 7 or 8 cm. Care should be taken to inject the solution along the proposed line of incision *intradermally,* following this with a second series of injections given subcutaneously and in fairly copious quantities.

The skin and subcutaneous tissues are now incised down to the sheath of the rectus muscle. The solution is then diluted to a half or a fourth of the original 1 per cent, and the rectus muscles infiltrated generously by inserting the needle underneath the sheath at intervals so that the anesthetized areas will overlap each other, still keeping in the line of the original skin incision.

The rectus sheath is now cut vertically and if the incision happens to be exactly in the median line, the two recti may be easily separated from each other by blunt dissection. But no time should be lost in trying to find the apposing margins of the recti muscles. Where this division does not present itself at once, the operator should without hesitation continue his vertical incision, either by sharp or blunt dissection, through one or the other of the muscles, adhering as close to the mid line as possible. Once past the recti (whether accomplished through separation or incision of one of the muscles) the edges of the wound are retracted and the shining surface of the fascia transversalis comes into view. It is well here to insert the needle a short distance through the fascia transversalis in the

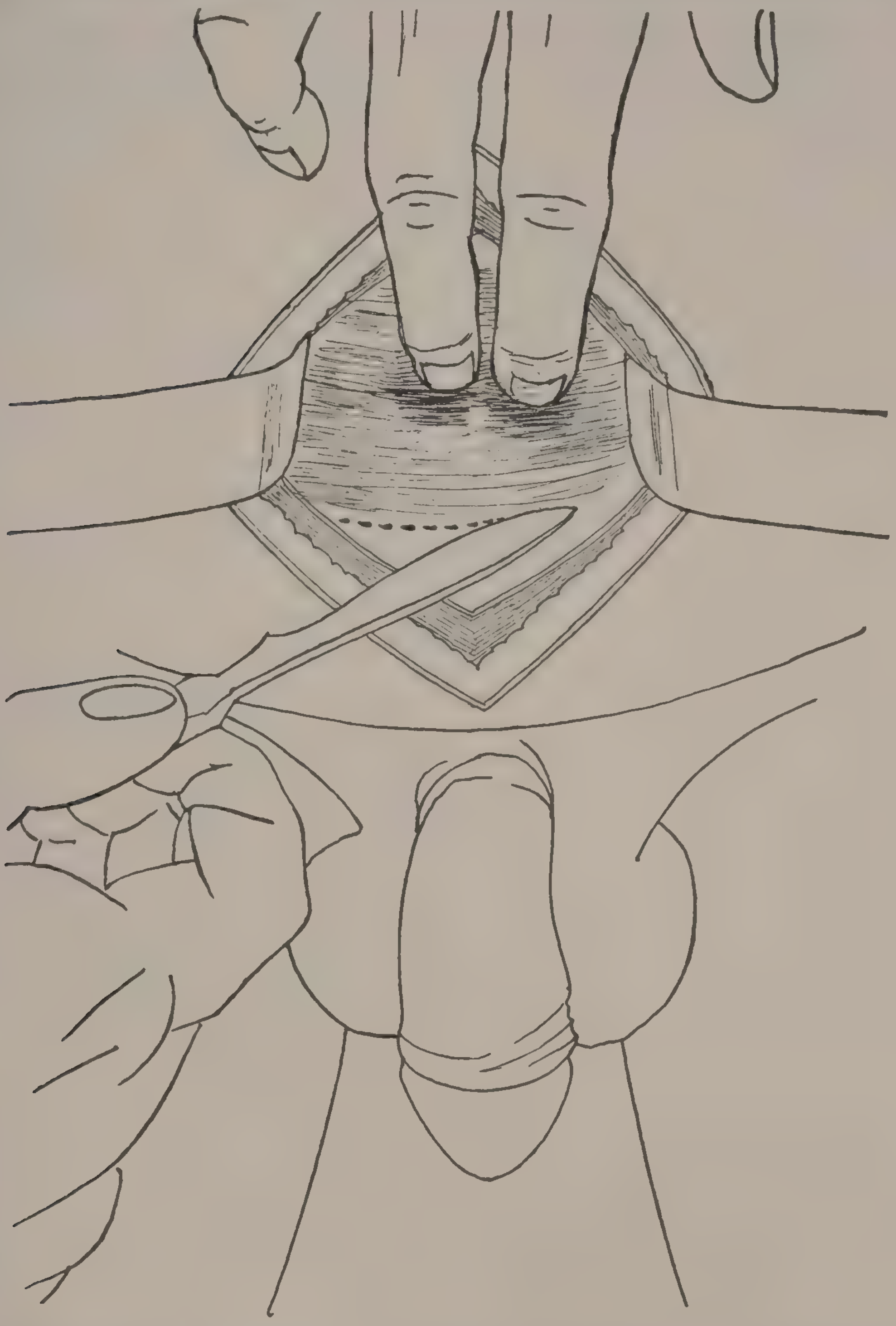


Fig. 1.—Exposure of the Bladder. Incision of the Fascia Transversalis. (Keyes.)

mid line just above the pubic bone and to inject several cubic centimeters of novocain (½ or ¼ per cent solution) into the loose tissues of the space of Retzius.

The next step, which is very important, but not difficult, is to cut transversely the fascia transversalis at a point just above and parallel to the upper margin of the pubic bone. By blunt dissection with gauze, the upper part of the fascia transversalis is now reflected upwards, carrying the peritoneum before it and thereby exposing the bladder. This part of the operation, which is done so readily under general anesthesia, is, with local anesthesia, likely to be attended by more or less pain. It therefore behooves the surgeon at this juncture to proceed with the utmost gentleness, and, if necessary, bolster up the patient's nerves with words of encouragement. If the fascia transversalis does not strip back readily, dissection with the scissors or knife may be used where required.

The bladder is readily recognized by its muscular walls, on which lie large veins radiating upward and outward from the pubis. Since it is desirable to open the bladder high up, the fascia transversalis is reflected back 6 centimeters or more above the pubis. The anterior wall of the bladder is now grasped by means of clamps (Allis preferably) on either side of the intended line of incision and the latter, which should be vertical and in an area not crossed by large veins, is infiltrated with novocain.

If the location of the tumor has been ascertained by previous examinations, such as cystoscopy or cystography, care should be exercised to avoid entering the bladder through the growth.

In order to prevent the possible dissemination of malignant tumor cells, before opening the bladder, gauze, soaked in 95 per cent alcohol, should be packed around the area where the incision in the bladder wall is to be made.

Steadying the bladder wall by means of the two clamps previously applied, an incision is made between them in the infiltrated line. The cutting is best done slowly in order to go just to the mucosa without penetrating it. When the latter is reached, it bulges up into the wound and resembles a small thin-walled cyst.

Unless the bladder be filled with air, a catheter should be inserted (provided the patient's condition will permit) and the bladder emptied. The clamps are then transferred to the mucosa and, with an aspirating tube in hand, the mucosa is opened by means of a short incision and the aspirator at once inserted. In this way, the bladder may frequently be emptied without spilling a drop of its contents about the wound. For the beginner, however, it may be simpler to open the bladder by introducing the knife through all coats at once by a sort of stabbing motion, but should the bladder contain fluid it is difficult to prevent the bladder contents from spilling over the wound.

In most instances, the clots are of such size that it becomes necessary to enlarge the bladder wound to facilitate their removal. This is best accomplished by irrigating with a hot sterile solution, such as normal saline, boric acid, potassium permanganate 1 : 3,000, acriflavine 1 : 5,000 or sterile water. The irrigating can is held high up above the wound so that with the tube introduced into the bladder the force will be sufficient to wash the clots out. Very large clots may be picked out with a sponge stick. By the time the bladder is free from clots, the active hemorrhage has usually ceased or else it is so slight that it can be expected to stop with proper drainage within a short time. But if the hemorrhage is at all profuse, the bleeding point should be located and the surface of the tumor seared with the actual cautery. To do this, it may be necessary to put the patient in the Trendelenburg position, open the bladder still wider and use retractors in order to expose the entire surface of the mucosa.

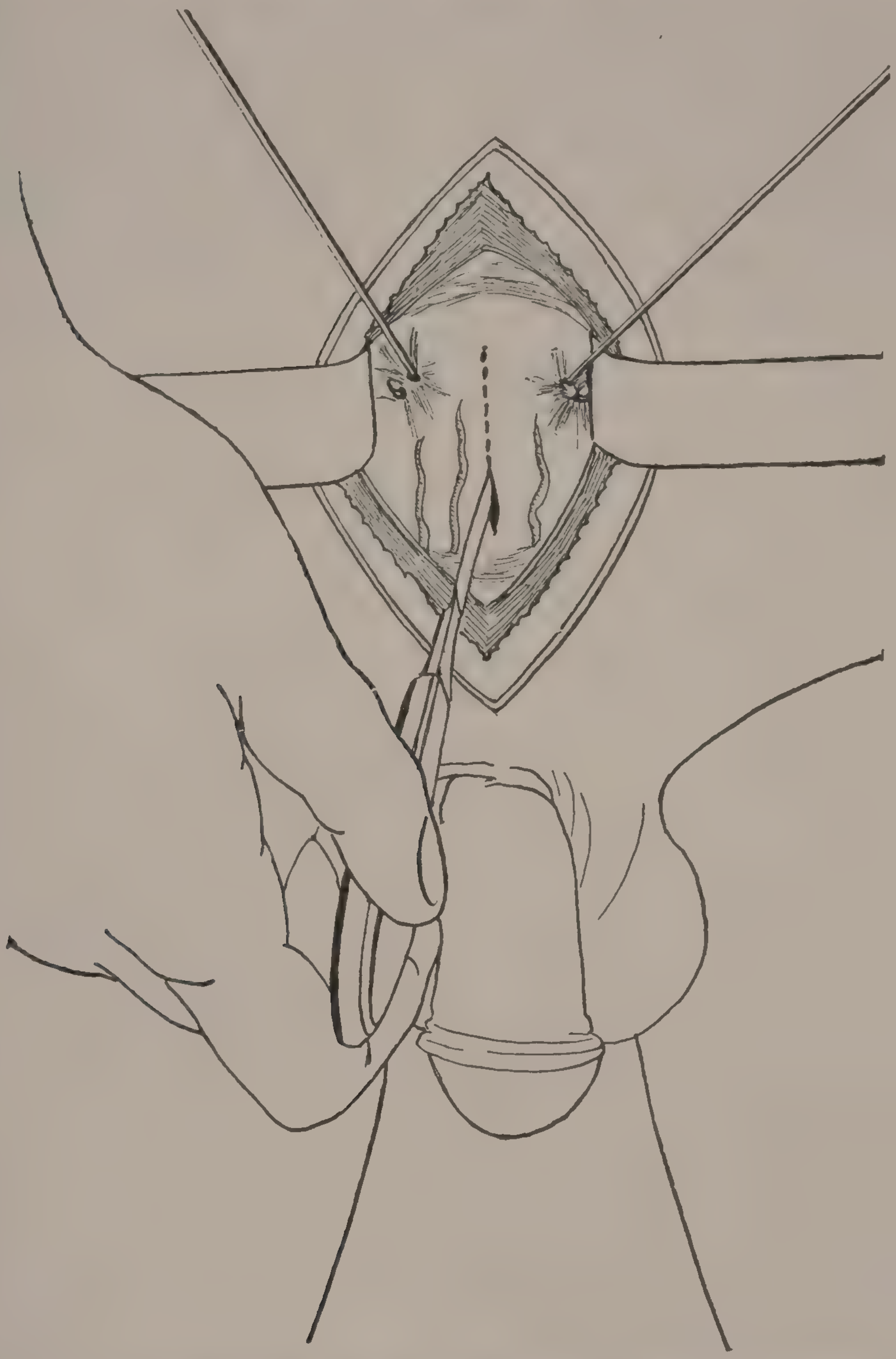


Fig. 2.—Incision of the Bladder Wall. The bladder is supported by Allis clamps or skin hooks. (Keyes.)

Should the bleeding arise from a pedunculated growth, which is easily accessible, the whole growth may be removed by cutting through the pedicle with the actual cautery.

If the bleeding cannot be controlled in any other way, packing the bladder with strip gauze about 2 inches in width may finally be resorted to. But it must be remembered that the gauze, a foreign body, may thwart the very end desired by setting up more or less violent bladder contractions. Hence, if it does not control the hemorrhage fairly promptly, it should be removed and drainage alone depended on.

Whether the hemorrhage is immediately controlled or not, the rubber tube selected for drainage should be large enough to permit the introduction of a small pair of sponge sticks, the latter to be used after the operation for the removal of those clots that will not pass voluntarily.

The drainage tube should be straight, open at both ends and contain one or two large eyes close to the end to go into the bladder. It is inserted just far enough so that the upper eye, if there are two, will be just below the bladder wall which is closed around it. This means that the length of tube inside the bladder will not be sufficient to rest against the bladder neck and cause contractions. Also there is less chance that the short tube will impinge upon a growth and cause hemorrhage.

The bladder is now closed so that the tube fits fairly tightly; interrupted sutures of plain No. 1 catgut, which include all coats, except the mucosa, are used. A purse-string suture may be placed in the bladder wall around the tube to hold the latter in place. A continuous suture of plain No. 1 catgut, is used to reënforce the first line of sutures, if the patient's condition will permit of the additional time required to do the suturing.

At this point the wound should be gently mopped off and either alcohol (95 per cent) or ether poured generously into the wound for the purpose of destroying malignant tumor cells.

The bladder is now anchored to the abdominal wall by a suture of plain No. 1 or No. 2 catgut on a curved needle, passed as follows: At a point about midway between the upper and lower ends of the abdominal incision, the needle is entered into the sheath of the rectus muscle and the muscle itself 1/4 inch from the line of incision. It is then carried through the coats of the bladder wall (mucosa excepted) just above the drainage tube, taking a bite about 1/2 inch wide. Care should be taken in this last maneuver to avoid the peritoneum. The suture is then entered into the rectus muscle of the opposite side 1/4 inch from the cut edge and out through the sheath. When this suture is tied, it pulls the bladder high up into the wound, and, by fixing it to the rectus muscle, makes the opening easy to find in case the drainage tube is accidentally pulled out during the postoperative period and must be reinserted.

The abdominal wall above the drainage tube is now closed, first by suturing the sheath of the rectus and then the skin. The part of the wound below the tube is *left wide open* in order to drain the space of Retzius, the loose areolar tissue of which invites infection. Three gutter drains, made by splitting rubber tubing lengthwise, are employed. Two are inserted laterally (one on each side of the bladder) so that they cross x-wise as they emerge, and the third is placed straight down behind the pubic bone.

Excision of Bladder Tumors and Destruction by Cautery.—If the growth lends itself by its size and position to excision, the mucosa which it involves may be the only part of the bladder wall removed or the entire section of bladder wall on which the growth rests may be excised wide of the tumor's margin. In dividing the tissues, as for example in cutting through the bladder wall, the actual cautery is used in preference to the knife, both to prevent hemorrhage and dissemination of tumor cells. Growths which are not amenable to resection may be attacked by radium plus the actual cautery, diathermy or the radioterm.

RADIATION

This term signifies treatment by radium (either the element itself or its emanation) and deep X-ray therapy, or a combination of radium and X-ray.

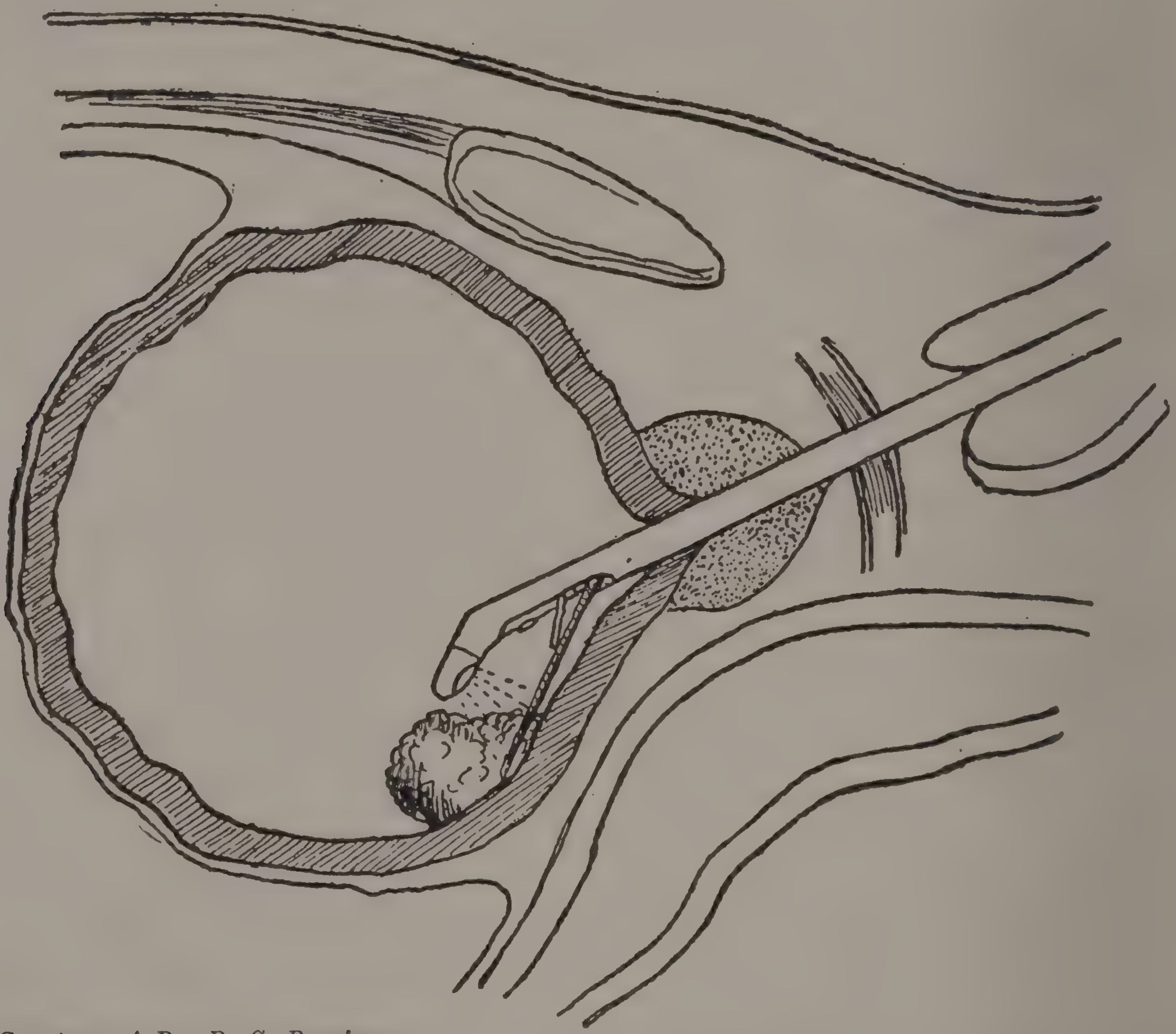
Radium Emanation.—On account of the comparative ease with which it may be obtained and the flexibility of its usage, radium emanation is preferred in most instances to the element. There are several reputable commercial houses that supply the emanation, accurately measured, together with instruments for its application or implantation.

Many growths in the bladder may be treated with radium emanation introduced through the cystoscope or urethroscope, while certain others require opening the bladder for its employment. In both instances either screened emanations or radium seeds are employed (see Volume I, Chapter XIII on Radium Therapy).

Technic in Cases Suitable for Cystoscopic or Urethroscopic Treatment.—If the tumor is relatively small and accessible to cystoscopic manipulation, radium seeds are implanted. This is done by means of a very narrow flexible tube carrying a short needle at one end. The instrument is chiefly constructed of wire similar to that employed in cystoscopic operating forceps and is used in the operating cystoscope.

The cystoscope is passed, the bladder washed, if necessary, and, after obtaining a good view of the tumor, the needle of the implanting instrument, previously loaded with a "seed," is introduced directly into the growth so that its point is well beneath the surface. The wire obturator, which ejects the seed, is then pushed home.

The needle is then withdrawn, and more seeds similarly implanted into other parts of the growth. The number of seeds to be employed depends of course on the dosage of emanation and the size of the growth. Ordinarily the rule employed by Barringer is the safe one to follow. He implants seeds containing 0.6 or 0.7 mc. of emanation so that there are approximately two such seeds to every cubic centimeter of tissue. If, however, one is at all in doubt about the size of the dose, it is better to err on the side of too little rather than too much and perhaps avoid a distressing radium burn. It is therefore oftentimes more prudent to use seeds of no greater strength than 0.5 mc.

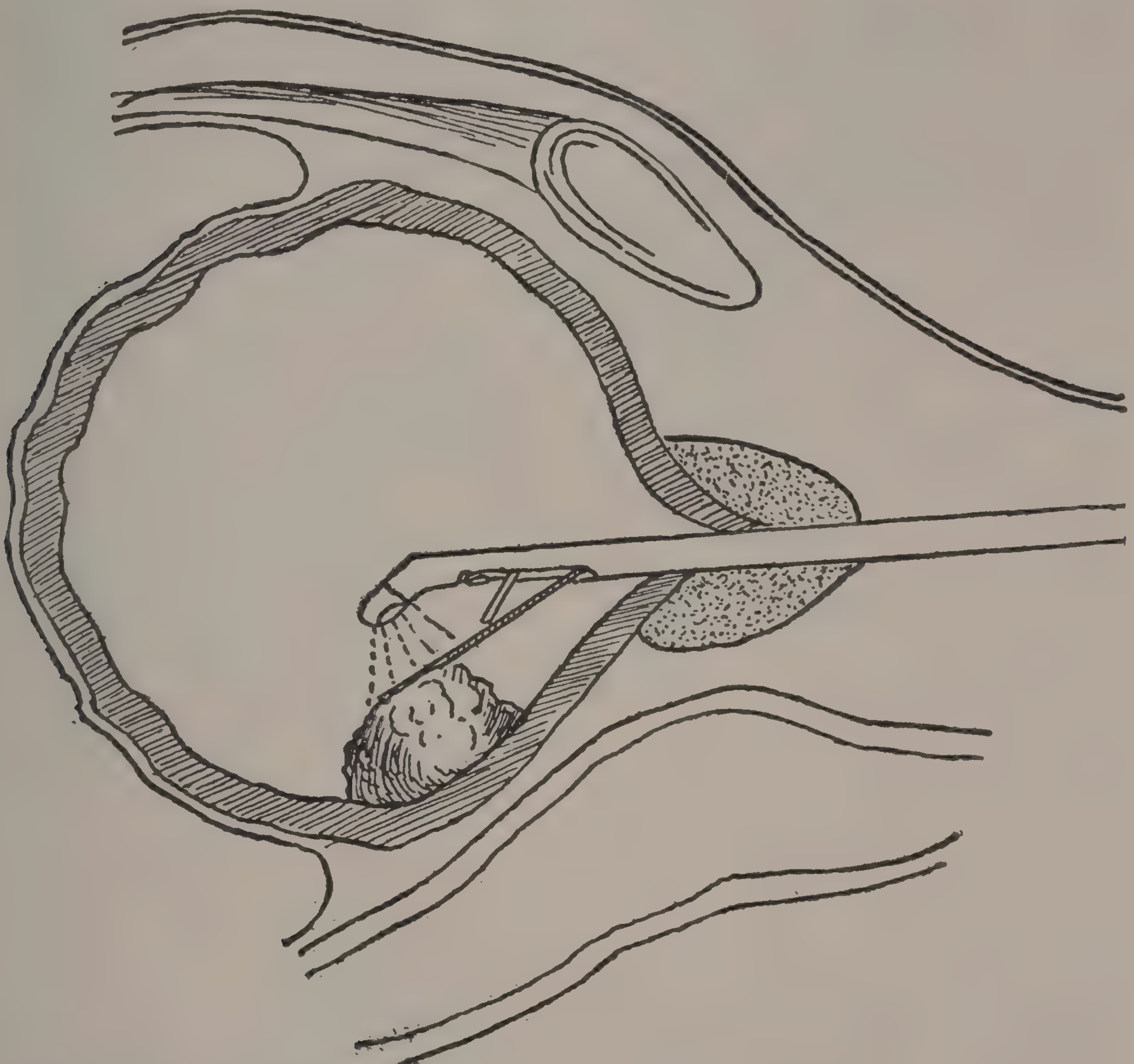


Courtesy of Dr. B. S. Barringer.

FIG. 3.—IMPLANTATION OF RADIUM "SEEDS" BY MEANS OF THE CYSTOSCOPE. By substituting the electrode for the radium applicator, this illustration also shows how a bladder tumor is attacked by fulguration.

The instrument mentioned above is also used in the surface application of radium by means of the cystoscope. Instead of the implanting needle, a larger needle containing a hundred or more millicuries of unscreened radium is substituted. This is held against the tumor under cystoscopic vision for a half hour or less. The operation may be repeated every two or three weeks, depending on the amount of discomfort the patient may suffer.

In instances where the growth is so situated that cystoscopic or urethroscopic approach for implantation is impossible, as, for example, a growth surrounding the bladder neck, surface application without the aid of the cystoscope may be employed. For this purpose the radium emanation, screened in a silver tube, whose walls are 0.5 mm. thick, is placed in a section of rubber tubing about 2 cm. long and ½ cm. in diameter. The walls of the latter are about 2 mm. in thickness and also serve as a screen. A stout cord, similar to the ordinary fishing line, is tied around the tubing near each end, thereby securely enclosing the silver tube. About 1 foot of cord is then fastened securely at one end of the rubber tube for the purpose of withdrawing it after the treatment is finished. Two such tubes containing from 25 to 100 mc. are usually employed. They are sterilized before use by boiling.

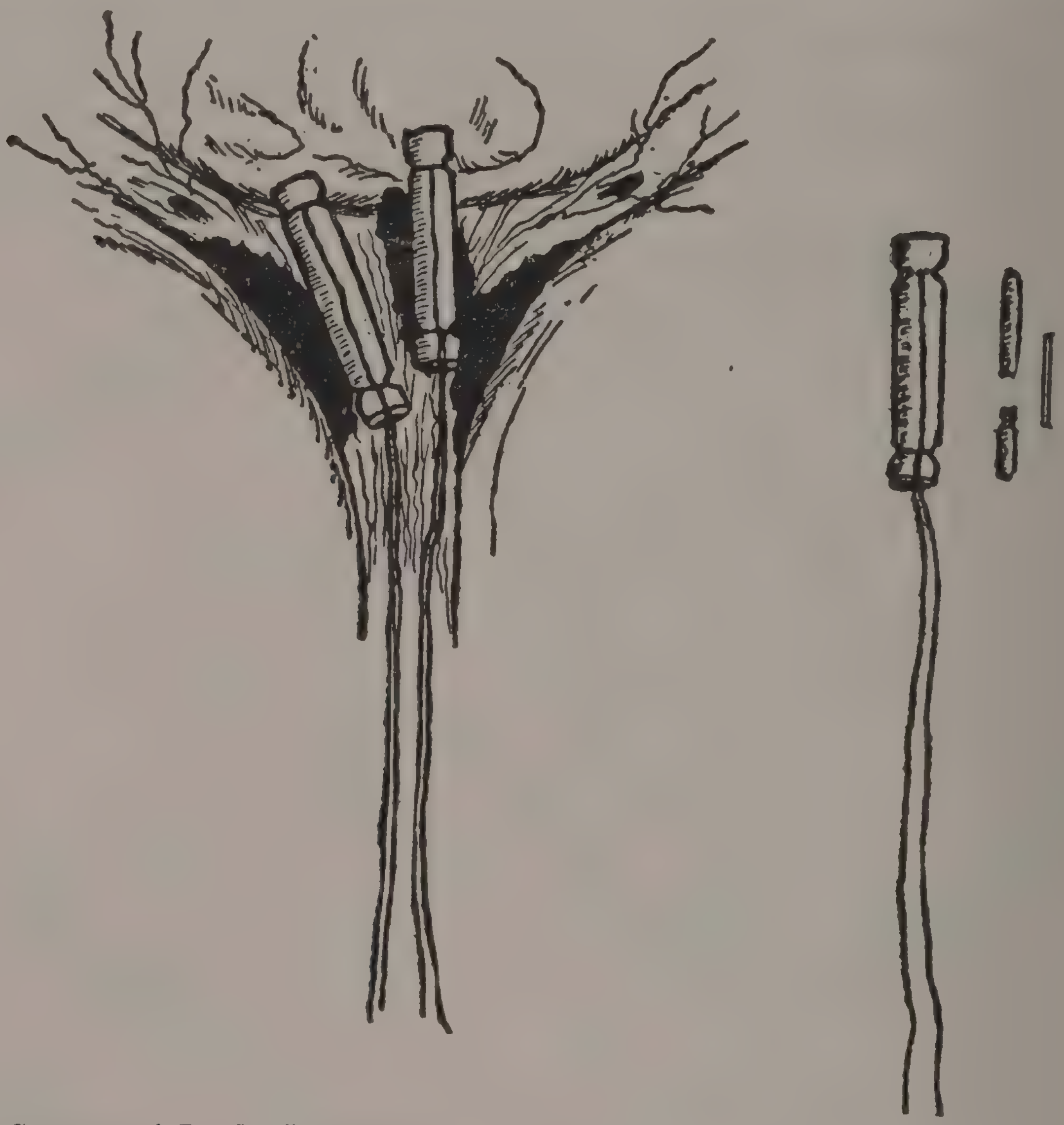


Courtesy of Dr. B. S. Barringer.

FIG. 4.—APPLICATION OF UNSCREENED RADIUM EMANATION TO SURFACE OF TUMOR BY MEANS OF THE CYSTOSCOPE.

Technic of Introducing the Tubes.—The instruments needed are an endoscopic sheath with obturator (the Geiringer and Young urethroscopes are both admirable for the purpose) and a straight metal rod about 10 inches long and ¼ inch in diameter. The endoscope is introduced into the bladder as if for urethroscopy and the obturator withdrawn. The bladder is then irrigated with sterile water and filled with about 6 ounces of solution, a finger being held over the end of the sheath to prevent the escape of the water. One of the tubes is lubricated with K-Y, placed in the open sheath and shoved into the bladder with the metal rod, care being taken that the end of the cord projects from the end of the sheath after introducing the tube to which it is attached.

The other tube is then introduced in a similar manner and the sheath immediately withdrawn. There should still be enough fluid left, when the second tube is introduced, to distend the bladder to some extent; otherwise it is difficult to introduce the tube.



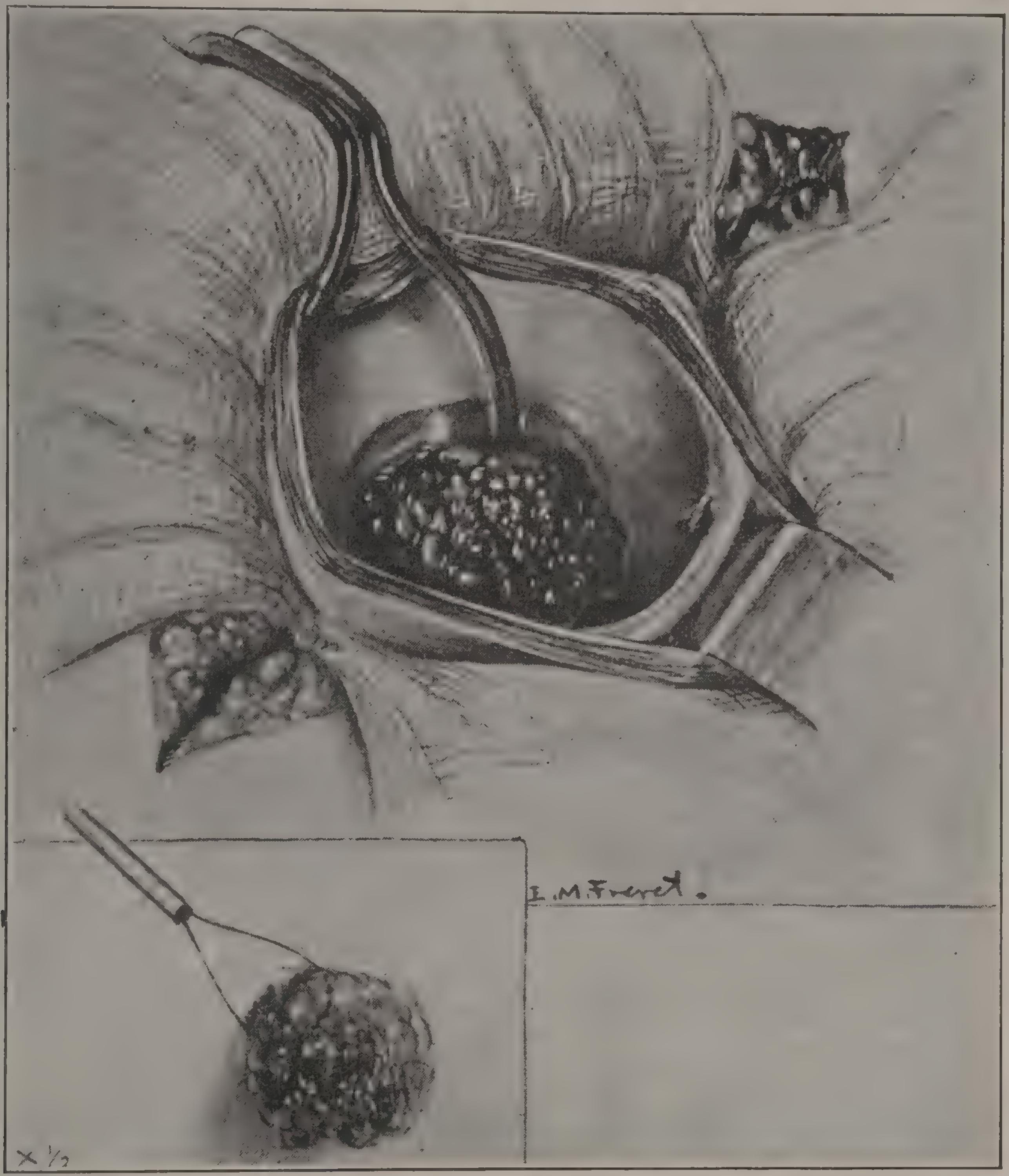
Courtesy of Dr. B. S. Barringer.

FIG. 5.—APPLICATION OF SCREENED RADIUM EMANATION TO GROWTH NEAR BLADDER NECK. The capillary tube containing the emanation is placed in a silver tube which is encased in a short rubber tube.

After both tubes are introduced, the remaining bladder fluid should not be allowed to gush out for fear of washing out one or both rubber tubes. Gentle traction on both cords, until resistance is felt, will pull the tubes down into the bladder neck where they should be. If desired, the latter maneuver may be done under vision by the introduction of a cystoscope, but care must be taken to avoid pushing the cords along with the instrument into the bladder.

After the procedure is completed, the cords are usually anchored to the thigh or perineum by means of adhesive plaster.

The length of time the emanation is allowed to remain in contact with the growth varies according to the strength of the dose and size of the growth. Ordinarily a dosage of 500 mc. hours is given. Therefore if two tubes of 50 mc. each are used, they would be allowed to remain in the bladder for five hours (50 by 2 by 5—a millicurie hour, being the number of millicuries of radium emanation times the number of hours it is employed in treatment).



Courtesy of Dr. B. S. Barringer.

Fig. 6.—Exposure of Tumor for Implantation of Radium "Seeds" Under Direct Vision. Insert shows Method of Snaring Off the Growth Before Implanting Radium Seeds in Its Base.

When the treatment is over, both tubes are withdrawn by pulling gently but firmly on the cords.

The patient is of course kept in bed during the treatment and, if he has pain, is given sedatives. If he cannot void, he is catheterized. If he is an ambulatory patient, he can probably attend to his regular duties by the following day.

The patient is usually cystoscoped at intervals of two weeks to note the effect of the treatment and if necessary the latter may, as a rule, be repeated in six weeks or two months.

Implantation of Radium Seeds after Exposure of the Growth by Suprapubic Cystotomy.—In cases where the growth is too large or otherwise unsuitable for the cystoscopic introduction of seeds, or where, in certain instances, the bladder is opened because of clots or retention, radium seeds are implanted under direct vision.

Especially constructed long needles with wire obturators which eject the seed are used for this purpose. Usually ten or twelve needles are necessary, because as each one is introduced into the growth it is allowed to remain in order to mark the spot where the seed has been deposited.

Technic.—The bladder is opened according to the technic employed in suprapubic cystotomy for drainage purposes.

With the patient in the Trendelenburg position, bladder retractors are used to bring the growth fully into view. If the growth is pedunculated, the major portion of it may be removed by means of the actual cautery or wire snare and seeds implanted in its base only. If it is the sessile type, however, and bleeding likely to be encouraged by manipulation, it is best to promptly implant the seeds without tampering with the growth.

To implant a seed, the needle is pushed firmly into the tissue until its eye is well beneath the surface (the depth depending on the thickness of the growth) and the obturator pushed home. The next seed is implanted in the same manner and so on until all the needles have been introduced in a given area, remembering to leave each needle in place after the ejection of the seed. All the needles are then withdrawn and reloaded. Another area for implantation is selected and the process is repeated. The rule for dosage here is the one laid down in the foregoing, namely, the employment of two seeds of 0.6 or 0.7 mc. of emanation (or less when in doubt) to each cubic centimeter of tumor tissue.

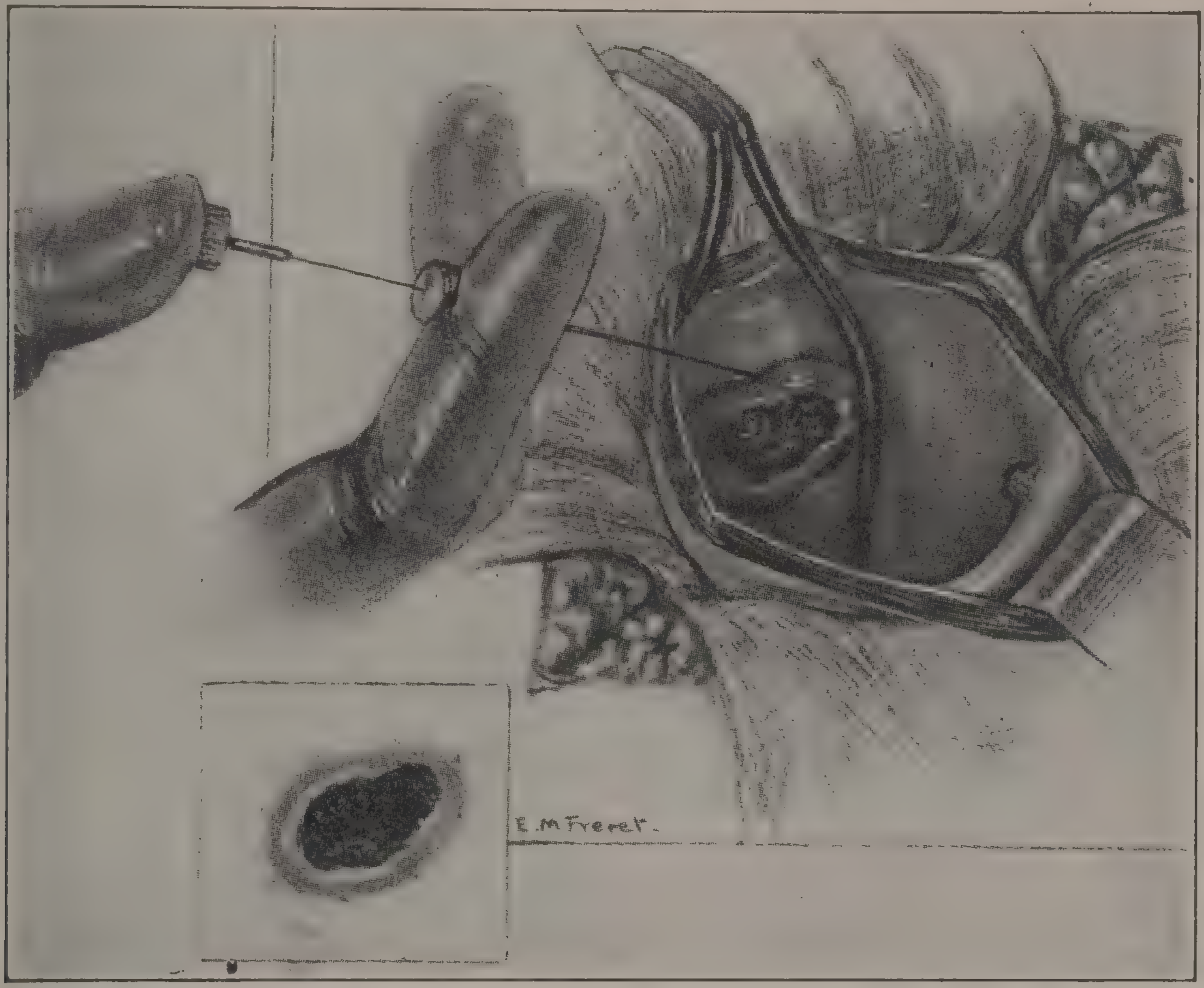
After completing the implanting operation the bladder is closed with drainage as in suprapubic cystotomy.

If the process must be repeated, it is usually not done until six months or a year later.

Deep X-ray Therapy.—This is said to be effective in certain types of bladder tumors, especially when used in connection with radium. Surprisingly good results are claimed by the champions of the method in cases that seem to offer absolutely no hope of improvement when treated otherwise.

Cystectomy, Ureteral Transplantation, Cystostomy.—There is hardly any excuse to-day for radical extirpation of the bladder. If the tumor continues to grow despite other forms of treatment, metastasis has probably taken place and little is to be gained except the relief afforded by the cessation of dysuria. But the latter end may be accomplished without subjecting the patient to such a major procedure by performing bilateral nephrostomy or transplanting the ureters. And since one or the other of these two measures is usually adopted as a preliminary stage to cystectomy, in the vast majority of instances it would be wiser to omit the latter altogether.

The indications for *ureteral transplantation* are: (1) involvement by the growth of the lower end of the ureter; and (2) a harassing dysuria which is frequently due to an intractable cystitis. In the former instance, the ureter is placed elsewhere to save the kidney; in the latter instance, transplantation is done to divert the urinary flow from the bladder. In either case nephrostomy will serve the same end.



Courtesy of Dr. B. S. Barringer.

FIG. 7.—IMPLANTATION OF RADIUM "SEEDS" AFTER EXPOSURE OF THE TUMOR (INFILTRATING CARCINOMA) BY SUPRAPUBIC CYSTOTOMY.

Cystostomy is performed chiefly for two reasons: (1) to provide a means of escape of urine from the bladder when there is obstruction at the bladder neck; and (2) to relieve the dysuria due to cystitis, the growth itself or both. It is preferable to ureteral transplantation for the relief of dysuria in that it is a simpler procedure, affords less chance of ascending kidney infection, and the resulting outlet for the urine is easier to take care of.

The technic is identical with that of suprapubic cystotomy with the addition that a suprapubic drainage tube, preferably of hard rubber or silver, is constantly kept in place to prevent the healing of the opening and thereby encourage the formation of a fistula. This tube should be removed every few days and cleansed thoroughly before reinsertion, to prevent its being plugged by incrustations of lime salts.

TREATMENT BEST SUITED TO A PARTICULAR TYPE OF GROWTH

According to Geraghty, who has had much experience with all the types of treatment described in the foregoing, including deep X-ray therapy, the best results are obtained as follows:

Tumors	Method of Treatment
Papillomata.	Fulguration—or a combination of fulguration and radium applied to the surface.
Superficial, non-infiltrating papillary carcinomata (broad-based, flat, often covering considerable areas).	Combination of deep X-ray and radium.
Circumscribed infiltrating carcinomata.	Resection.
Extensive infiltrating carcinomata, which are inoperable.	Radium implantations.

Cysts for the most part are destroyed by fulguration. The smaller ones sometimes disappear after puncture with the operating cystoscopic scissors.

The other tumors mentioned in the beginning of the chapter are so rare that a discussion of their treatment is hardly worth while.

REFERENCES

Geraghty: Personal Communication.
Keyes: Urology, 1923.

OCCUPATIONAL DISEASES

CHAPTER XXXIII

INDUSTRIAL INFECTIONS

W. GILMAN THOMPSON

COMPARISON OF INDUSTRIAL AND OTHER INFECTIONS

The industrial infections, that is, infections acquired as hazards incident to industrial work, do not differ pathologically in any marked degree from infections otherwise originating. It is therefore unnecessary in this article to dwell in extensive detail upon the fundamental pathology of traumatic infection, but reference will be made to certain slight differences, which are as follows:

In workingmen, the skin of the hands and arms, and sometimes of the feet, is often considerably hardened through occupation, and the "horny hand" of the laborer offers a superficial covering which, on the one hand, may protect against external injury, and, on the other, when such injury has occurred, presents a toughened layer under which pus, once formed, is liable to accumulate in considerable quantity. In some instances the occupation of the employee itself may present special hazard of infection, in that the substances handled may already be infected.

Certain materials are well known to be infected frequently with septic germs and consequently those whose work necessitates handling of such materials are constantly liable to septic infection whenever they acquire even a slight wound. Such, for example, are men employed in industries dealing with dead animals and materials derived from them. Among these may be enumerated butchers, meat packers and canners, and those employed in handling garbage and various forms of fertilizer, both natural and artificial; also workers in feathers or hair, glue makers, wool sorters, and paper makers where woolen rags are used, soap makers, and tallow refiners. In most instances, however, it is the skin of the workman himself which harbors the germs of infection, which merely are given an accidental port of entry to the system through a wound or abrasion.

The primary infection quite often is complicated through the penetration of dirt into the wound. Particularly is this true among those workmen who handle oily and greasy substances or tenacious materials such as glue, wax, resin and tar.

In other cases, as, for example, in dealing with bronze powders, more or less irritant metallic substances may be conveyed into a deep punctured wound together with the germs of infection. The irritation caused by such an apparently simple substance as iron rust may complicate the situation when deeply imbedded in or beneath the skin. Moreover, rust may form around imbedded particles of iron or steel. Particularly is this the case when such particles penetrate the cornea, where rust sometimes forms with extraordinary rapidity. In such instances the foreign body complicates and serves to intensify the infection proper.

All the above-mentioned conditions, however, result in no essentially peculiar pathology, but merely accentuate the well-known picture of traumatic infection.

Neglect and ignorance on the part of the workingman, or fear of losing time by quitting work, too often complicate the situation by enabling the infection to gain several days' headway before treatment is sought. It thus may transpire that infection of a finger, for example, which readily might have been controlled at the outset, is permitted to involve the entire hand or extend through the lymphatics of the arm to the axillary glands, causing cellulitis, lymphangitis, and sometimes boils and carbuncles. In such cases extensive incision may ultimately be required to lessen tension and evacuate pus, with subsequent deforming contractures of scar tissue and prolonged or even permanent disability. The writer has seen several cases of necrosis of the phalanges necessitating amputation which followed neglect of primary slight infections of fingers or toes. Periostitis and localized osteomyelitis may result. In fact, cases are recorded where systemic infection has followed with septicemia and death.

The serious wound, such as extensive laceration from being caught in machinery, or the compound fracture received in falling from a scaffolding, commands immediate surgical relief so that infection may be dealt with at once; but the minor infections resulting from comparatively trivial abrasions or punctures are usually made light of by the hardy workman, accustomed as he is to knocks and blows, and it is this type of neglected infection which is responsible for so much "lost time" in many industrial plants.

EXTRAHAZARDOUS INDUSTRIES

Certain industries from their nature are extrahazardous from the standpoint of minor infections. Foremost among them are those in which sharp cutting tools are much used, as, for example, in cutting the sheets of thin rubber from which rubber gloves, inner tubes of automobile tires and many other rubber articles are made; the manufacture of much hardware and cutlery; the handling of sharp sheets of metal, as in making tin cans; the use of sharp knives or scrapers, by butchers, tanners, furriers and felt hat and shoddy makers; the handling of rough material from which splinters are derived, as in wooden box and barrel making; sawmill work; work involving the driving or pulling of nails, etc.

In all forms of carpentry, but particularly in destructive work, such as demolishing buildings, splitting up old boxes, barrels, etc., and remaking them, there is the hazard of puncture wounds or abrasions from contact with wooden splinters, rusty nails and similar sharp substances. In much minor cutting machinery and lathe work and in poorly protected stamping machines there is constant liability of crushing the fingers, and such accidents quite often involve infection. Die cutters are particularly liable to this trouble.

In the different forms of wire industries, as in making wire and wire cables, there is always the possibility of puncture wounds being acquired. Such wounds frequently are very narrow but quite deep, and, as in the case of fine nails, rusty particles are conveyed to the bottom of the wound. The manufacture of wire brushes is particularly hazardous in this regard. In the opening of clam and oyster shells, infected wounds are common, being derived both from the knives used and the sharp edges of the shells.

Such are a few of the industries in which punctured wounds are common. Another group of industries includes those in which abrasions afford the site of infection. Such in particular are those in which the hands, and sometimes the feet, become more or less macerated and the integument eroded through prolonged soaking in fluids, such as dyestuffs or through contact with chemicals as in handling lime, lye, strong acids and alkalis, etc. In still other cases, dust, grease, oil and similar irritants tend to roughen the skin as in cement workers, in employees in oil refineries, in tar and turpentine workers, and the like.

A few only of the multitudinous possibilities of industrial infections may be mentioned here more in detail:

As illustrating an unusual method of acquiring infection, the following incident has been reported to the writer. A diver working over a pipe line at considerable depth below the water's surface scratched his hand seriously upon a barnacle. The wound became infected, and the man neglected it until a very serious septic condition had arisen. In a case of this type coming up for compensation it would be difficult to produce a witness to the accident unless mermaids were present!

In the oil-producing industry, long wire cables to which plummets are attached are used in sounding the wells. The plummets, while being dropped through the vertical pipes, are handled by the workmen, the wire cables slipping through their hands. These cables, when slightly worn, become frayed and, as they slip through the hands, puncture wounds are made by the wire ends. As the workmen's hands are always begrimed, this is a very common source of infection. The punctures are narrow and often deep, and give rise to considerable trouble.

Another common source of infection arises through the splashing of acid used in a great variety of industries, as in tinning processes. The drops of acid falling anywhere on the surface of the body, or the hands or feet, cause small burns, followed by more or less slight sloughing. A day or two after such burns have been received, they are very liable to become infected.

An interesting difference between men and women employees was reported to the writer by the company doctor of a large oil refinery where extensive manufacture and filling of tin cans was conducted. The hands of women as a rule have more delicate skin, that is, they are not so subject to callous formation as those of men. They, therefore, are more sensitive to the slight burns from splashing of acid droplets, which are more promptly washed off, with less resulting infection. The men, on the other hand, do not always notice such slight acid burns, and any infection beneath a calloused surface is imprisoned for a longer time than it otherwise would be, and hence pus accumulates in considerable quantity before its presence is recognized.

Another illustration of this latter topic is the following: A number of ignorant peasants, not used to handling sharp spades, were set to work digging trenches for a pipe line in some hard ground. Not being expert in handling spades, instead of pushing them in with their feet, they pounded them in with repeated blows of the feet, thereby, after a time, seriously bruising them. The soles were dirty and calloused, and readily became infected when serious abscesses formed beneath the hardened integument.

Sailors employed on vessels used in the transportation of oils, wax, and greasy substances of various kinds are exposed to the contact of these substances with the skin, the pores of which become choked, and the hands and feet may become covered with callosities, and readily are infected when injured.

In addition to acid burns, the blisters caused by contact with hot metals, molten solder, etc., often become infected.

PREVENTION

The prevention of infection of cuts, punctures, abrasions and burns constitutes one of the most important problems for every industrial surgeon. Infection arising through acid burns sometimes may be prevented by the use of stout canvas or leather gloves, where the splashing of acid droplets is feared. But the wearing of stout gloves affords little protection from puncture wounds. As a matter of fact, it is difficult to get workmen to wear gloves, for their hands perspire, and if they become wetted in any way the wearing of gloves tends to cause maceration of the superficial layers of the skin, rendering it more sensitive to abrasion.

Employees should be given strict rules never to disregard open wounds, however trivial they may appear, and, if possible, be penalized for disregarding them. Foremen, particularly, should be instructed in the economic loss arising from neglected infections. Many industrial plants supply first-aid kits equipped with antiseptic dressings and bandages, but, where a first-aid station is reasonably near, supplying a company doctor or nurse, it is preferable to insist that the employee shall go to it at once when slightly wounded. In fact, some companies which at first supplied emergency kits have taken them out the better to emphasize the resort to dressing stations. Much, naturally, must depend upon the nature of the work, and the size of the plant. In field work, as in the oil-producing industry, pipe laying, telegraph and telephone-pole work, railway construction, coal and other mining, portable kits obviously are essential, and they must be equipped with antiseptic dressings which are both portable and stable. Tincture of iodin, for example, if supplied in such outfits, should be in form of ampules. As a rule, therefore, sterilized dressings, ready made, such as moist bichlorid gauze, are all that really are necessary for the first-aid kit, together with an assortment of sterile compressed rolled bandages adapted in different sizes for any portion of the extremities. Compressed cotton wool may be added for padding over the more extensive injuries, and a spool of rubber adhesive plaster is convenient to aid in holding compresses in place or fastening bandages where they are liable to slip. Each kit, moreover, should contain printed directions for the employee, for the most difficult thing to impress upon him is that the small wound should be cleansed from within, rather than from without, that is, by a free outward flow of blood, and not by mopping or rubbing with soiled handkerchiefs or rags.

In some cases, however, where an extensive abrasion has been covered over by contact with oil or grease, or by falling over gravel and dirt, it may preferably first be cleaned with pure hot water (which encourages bleeding), if obtainable, or with absorbent cotton soaked in gasolene, which is now almost universally obtainable. Small abrasions may be painted with a sterile cotton swab dipped in a 2 per cent solution of iodin. (The official tincture is 7 per cent.) Small cut or punctured wounds need not be painted with iodin, but allowed to bleed a little, in order that the issuing blood may carry out germs and dirt particles; but if complicated by an abrasion the surface may be painted before the sterile dressing is applied. The employee must be instructed that continued bleeding is only to be checked by continued pressure with a sterile compress, never by intermittent "mopping."

The instructions issued in a leaflet to accompany each emergency kit are thus formulated by the Factory Department of the British Home Office:

"A Scratch or Slight Wound:

"Do not touch it.

"Do not bandage or wipe it with a handkerchief or rag of any kind.

"Do not wash it.

"Allow the blood to dry and so close the wound naturally; then apply a sterilized dressing and bandage.

"If bleeding does not stop, apply a sterilized dressing and sterilized wool, then bandage firmly.

"If the wound is soiled with road dirt or other foul matter, swab freely with wool soaked in the iodin solution (2 per cent) and allow the wound to dry before applying a sterilized dressing."

Where wooden splinters or sharp fragments of metal have penetrated deeply into the fingers or toes, they may often be clearly seen by placing a strong ordinary flashlight behind the finger or toe, which renders it sufficiently translucent to enable detection of the foreign body.

The hands and feet and various parts of the bodies of workingmen are quite often saturated with oil, grease, or dirt adherent to the skin. When a puncture wound, cut or abrasion occurs through such skin, it may be cleansed by the use of gasoline, followed by tincture of green soap in hot water.

When infection is feared from a deep puncture wound, it is often best immediately to enlarge the orifice of the wound, to enable reaching the bottom with antiseptics, and, subsequently, to apply sterile dressings and secure proper drainage. The wound may be probed with a sterilized cotton swab dipped in some antiseptic. For this purpose dichloramin-T, full strength, may be used, and immediately washed out with warm water. A small wick of gauze may be left in the wound, should it be deep enough to require drainage. It is important to keep this gauze moist so as to act as a wick, and not to let it dry and act as a cork with pus pent up beneath it.

The choice of antiseptics to be used as preventative of local infection includes a very wide range of substances, and each surgeon is likely to have his own ideas regarding their selection and mode of application. In a recent conference of twenty industrial surgeons at which this matter was under discussion, it was interesting to note that there were as many different opinions presented. As a matter of fact, there are a large number of antiseptics which have long proved almost equally efficacious, and there is more or less fashion regarding their use, some being in vogue for a time, and others taking their place.

As a general proposition, when the wound is fresh, and as soon as possible after it has been received, wet applications are indicated. These at first may be used in full strength, and subsequently in weaker solution. Later on, as healing progresses, various ointments and powders may be applied. In only quite superficial wounds, such as slight abrasions, should the latter type of antiseptic first be employed.

In the early stages of superficial infections, while active secretion is going on in the wound, and where more or less extensive gauze or similar dressings have been applied, it is most important to keep these dressings continuously moist. The patient should be cautioned to do this and, particularly at night before retiring, to moisten the dressings with some antiseptic solution or with plain water recently boiled.

Among the fluid antiseptics which are at present in vogue may be mentioned: dichloramin-T, with an initial application of 2 per cent in strength, and subsequent application of .5 per cent; saturated boric acid solution; alcohol; hychlorite; tincture of iodin, and also Churchill's tincture; cresol 2 per cent; picric acid, in aqueous solution in strength of .5 per cent; bichlorid of mercury solution 1 to 1,500; and hydrogen peroxid. Of these, hychlorite relieves pain more than many other antiseptics, used in strength of 1 ounce to 2 quarts of water. The powders which often are used, either in pulverized form or made into ointment, comprise such antiseptics as zinc oxid, iodoform, boric acid and aristol.

Where there is chronic pus discharge, as from a fistula, or otherwise retarded healing, balsam of Peru with castor oil makes an excellent dressing, and iodoform, and particularly aristol, make good applications.

The objection to oily and creamy dressings, as often used, is that unless frequently supervised by the surgeon they are liable to become dry and harden, forming undesirable crusts under which pus may accumulate.

In cases where very tough callus interferes with the free evacuation of pus beneath, it may be softened by the use of a solution consisting of 60 per cent alcohol with 40 per cent glycerin applied on compresses.

The healing of contusions with abrasions over the skin is often retarded by the movements of the tendons beneath. It is sometimes useful to paint the edges around an abrasion with iodin, without painting the open wound itself.

When scabs form over infected superficial burns, they may be removed by soaking under oil compresses, and then dusted with aristol.

There follows a summary of a very simple method of dealing with slight abrasions or penetrating wounds which are covered with grease or dirt:

1. Cleanse with absorbent cotton soaked in gasoline (for abrasions).
2. Wipe with an applicator dipped in gasoline (for cuts or punctures).
3. Paint with an absorbent cotton swab dipped in 2 per cent iodin solution.
4. Apply a sterilized gauze compress.
5. Bandage the wound over the compress and fasten the bandage either by tying or with strips of adhesive plaster.

Among the merits claimed for the different antiseptics above numerated, none has given rise to more discussion than iodin. Although probably 80 per cent of the industrial surgeons in this country favor the use of iodin, there are some who discard it entirely. The British Factory Department of the Home Office recommends a standard strength of 2 per cent iodin in alcohol solution. A decided advantage in the use of iodin solution is that it eliminates the necessity of washing the wound, which, if done at all, requires care and skill. It also is hemostatic. Collis and Greenwood state in *The Health of the Industrial Worker* that "elimination of washing and the use of sterilized dressings alone reduced septic cases by some 80 per cent when introduced in a group of engineering factories." And Mock states that "the immediate use of tincture of iodin to an injured part by the employee himself or by a fellow employee is the most important first-aid procedure which can be adopted in industry." He claims a reduction of 28 per cent of infections the first month after this procedure was installed in his service. An objection to the use of iodin is that it conceals hyperemia around the wound, and, if its use be continued, or if it be employed in full 7 per cent strength of the official tincture, it may retard healing. It is quite common for private patients to keep a bottle of tincture of iodin in the house for the purpose of touching such cuts and abrasions as made by needles, scissors, penknives, etc. If the tincture of iodin is kept too long, the cork in the bottle becomes destroyed, and more or less alcohol evaporates, thereby making the solution far too strong.

As previously stated, the main difference in the so-called industrial infections as compared with ordinary wounds is a matter of degree rather than of kind. The workman who has received a slight cut or abrasion regards it as of no consequence, or fears, if he reports it, that it may mean time and wages lost; and it is only after two or three days, perhaps, when the throbbing of a felon begins to give him pain, that he seeks relief. Moreover, he probably has had many slight cuts and abrasions from time to time which did not happen to result in any serious infection, and he cannot be made to realize that it was only through good fortune that he escaped what should be regarded as a genuine menace.

Experience, however, shows that, in industries in which cuts and abrasions are a frequent hazard, strict enforcement of the rules of the medical department in regard to dealing with them reduces enormously the possible days of disability, with resultant great economic saving. Frequent "first-aid" talks by the doctor may do much toward helping to achieve the best prophylactic measures against infection.

CHAPTER XXXIV

INDUSTRIAL INTOXICATIONS

JAMES FREDERICK ROGERS

Introduction.—That serious poisoning can occur from materials with which men and women come in contact in various occupations, has been recognized for a long time. Some of the best known of these toxic agents still harvest a hideous yearly toll of victims, while progress in chemistry has added many new poisons from which the unprotected and uninformed worker may suffer injury or death. Such substances are not only increasing in number, but some of them are used in such a variety of industries that cases of intoxication often find their way to the physician from unexpected sources.

Entry and Exit.—Industrial poisons may take the form of fumes, dust, or liquid, and find entrance to the body either by way of the respiratory passages, the digestive tract, or through the skin. While toxic material in the form of dust is taken in by respiratory movements, much of it is swallowed and finds actual admission through the walls of the stomach or intestine. Many substances affect the skin locally, but comparatively few produce systemic effects through this channel.

Poisons, changed or unchanged, leave the body chiefly by way of the kidneys, the alimentary tract, the lungs, or the skin. Where the rate of elimination is less than that of intake, some toxic materials are stored in various organs and may remain in the body long after the workman has abandoned his dangerous occupation.

Pathology and Symptoms.—Poisons derived through occupation do not differ in their effect from poisons acquired under other circumstances. Each poison has a more or less selective action, producing more or less characteristic effects. The symptoms displayed depend also, to some extent, on the portal of entry as well as upon the size and frequency of the dose.

Acute poisoning, produced by comparatively large and overwhelming amounts of the intoxicant, result in chemical changes in the tissues, usually of a transient nature, while in chronic cases the frequent intake of small quantities results in slow organic changes, the recovery from which may be slight, even after removal from the source of injury.

As for bacterial intoxication, there is a great variation in individual susceptibility. Moreover, the effects depend on age and sex, the immature being more sensitive than the adult, and the female sex often less resistant to certain substances. Alcohol has a decided influence in increasing susceptibility to some poisons, and poor nutrition tends to lower resistance.

The clinical picture of poisoning by the same substance may differ greatly in different subjects; partly, no doubt, through greater vulnerability of certain tissues in one individual than in another, but also because the results in one case may be due not only to the poison under suspicion but to the admixture of other poisons present as impurities. In some of these mixed intoxications the effect of one substance may be modified, neutralized, or heightened by another. Not infrequently a worker may be exposed to a number of dangerous agents, and the physician must be satisfied with a diagnosis of industrial poisoning, the particular substance giving rise to the symptoms being in doubt.

Diagnosis.—The occupation of a patient is one of the most important matters to be inquired into in any case of illness, and the physician who probes deepest for such information will make fewest mistakes in diagnosis. He will not be content with learning that a man is a wood worker, or that a woman is employed in making artificial flowers. The former is, in the great majority of cases, free from special industrial danger, but the case in hand may be suffering from a weakness of the heart and general depression from working in boxwood. Obscure symptoms appearing in the maker of flowers may be due to the action of lead from which her patterns have been cut. So frequent a symptom as colic, of so common an industrial poison as lead, has been too frequently misinterpreted for want of careful inquiry into occupation, and, in at least two such cases, four laparotomies were performed on each of the victims before the true cause of the complaint was discovered.[1]

Prevention.—From every point of view the importance of prevention of industrial poisoning is evident, though we have been slow in putting such obvious truth into practice.

The substitution of a non-toxic substance for a poisonous one is the ideal means of prophylaxis, and a striking example of results has been the practical abolition of phosphorus poisoning by the use, in the making of matches, of red phosphorus or the sesquisulphid instead of white phosphorus. Where such substitution cannot be effected the attenuation of the toxic agent by adequate ventilation will bring about a great reduction in occupational risk, while the supplying of protective clothing adds to safety.

It is obviously unfair to employ a man or woman in any hazardous work without informing them fully as to the possible dangers and how they are to be avoided. Moreover, a medical examination should be made at suitable intervals; those who are especially susceptible to the poisons should be placed at other work, and every effort made to maintain the health of those exposed.

---

[1] A valuable list of occupations with the poisons to which the worker may be exposed will be found in Bulletin No. 306 of the U. S. Bureau of Labor Statistics.

Treatment.—The treatment of acute cases consists chiefly in their prompt removal from the scene of intoxication and the application of the most effective means of helping the body to rid itself of the injurious agent. In chronic cases treatment consists chiefly in removal from exposure, relief of symptoms, and good hygiene. The wrecks from long-continued industrial poisoning are often pitiable indeed. To check the progress of the disease, or effect relief of symptoms, they must be removed from the source of their livelihood, and usually being unfitted through age, habits, or disability to take up another skilled calling, they often gravitate back to the old employment under the same hazardous conditions and go from bad to worse.

METALS AND THEIR COMPOUNDS

Antimony

Poisoning from antimony is said to occur among handlers of ore, polishers of steel, paint makers and workers in the chemical industries. It may help to complicate the picture of poisoning from other metals with which it is often present, and some of the chronic ailments of printers have been attributed to the presence of antimony in the type which they handle.

Pathology.—Antimony, or its compounds, enters the body in the form of fumes or dust by way of the respiratory or the digestive tracts. No cellular changes have been ascribed to its action except a moderate anemia, a reduction in the number of leukocytes and an eosinophilia—the proportion of these cells running from 10 to 25 per cent.

Symptoms.—In the acute cases attributed to this substance, there has been inflammation of the nose, mouth, throat, and bronchi, and disturbance of the digestive tract with vomiting, colic, and diarrhea. In severe cases, dizziness, faintness, and palpitation have been complained of, and albumin has appeared in the urine.

In chronic poisoning there are digestive disturbances, headache, dizziness, feeling of oppression in the chest, fatigue, irritability, nervousness, and insomnia. An itching dermatitis, which may become pustular, is frequently present.

The blood shows some decrease in red and white cells and a considerable increase in proportion of eosinophils. Antimony may be found in the feces.

Prevention.—Adequate means for the removal of fumes and dust should be installed and kept in operation. The skin should be protected from dust as far as possible by suitable work clothes. There should be the utmost cleanliness before eating, and a bath should be taken and the clothing changed after leaving work. Facilities for eating where there will be freedom from fumes or dust should be furnished.

Treatment.—The patient should be removed from exposure to the poison, a laxative given, and efforts made to improve his nutrition.

ARSENIC

Arsenic is used in a great variety of occupations and is also present as an impurity in many processes in which it is non-essential. The element is considered harmless, but intoxication may occur from the inhalation of fumes of arseniuretted hydrogen or from the dust of other compounds absorbed through the digestive or respiratory organs or through the skin. There is great difference in susceptibility to the poison and apparent immunity in some cases.

Pathology.—Arsenic has an inflammatory action on the mucous membranes and the skin. Absorbed in quantity, it causes destruction of the red blood-cells. Change in other organs is largely due to this hemolysis. The spleen is enlarged, there is fatty degeneration of the liver and kidneys, and the tubules of the latter are clogged with the remains of broken-down erythrocytes. The organs and fluids are stained with the blood pigments. The alimentary canal is congested and corroded. The symptoms are largely referable to the destruction of the red blood-cells and the consequent asphyxia of the tissues.

Symptoms of Acute Poisoning.—In the acute form, due to inhalation of concentrated fumes of arseniuretted hydrogen, the symptoms usually come on within a few hours. There is nausea, vomiting, abdominal pain and distention, diarrhea, frequent urination, drowsiness, weakness, dyspnea on exertion, and paresthesias of the limbs. In about twenty-four hours the urine becomes discolored with products of blood pigments. Fatal cases last from four to eight days, and a prominent late symptom is suppression of urine, probably from blocking of the tubules with damaged blood-cells.

The patient's face and eyelids are puffy, and there is jaundice, though not of a deep hue. The pulse is weak and rapid and the respirations increased in frequency. There are no objective nervous symptoms. A blood examination shows the red cells reduced—in one case to 2,000,000; the hemoglobin is correspondingly diminished, the index being little changed; the white blood-corpuscles are of the usual number and there is nothing significant in the differential count. The urine contains albumin and is stained with blood pigment.

Chronic Poisoning.—In cases of chronic poisoning the gastro-intestinal symptoms are prominent. There is nausea, vomiting, epigastric discomfort, and sometimes diarrhea. The nutrition suffers and the patient becomes weak. Multiple neuritis may appear and paralyses have occurred. In severe cases the central nervous system is affected and convulsions may occur. There is often dryness or soreness of the mouth and throat, and there may be a cough. A troublesome eruption of the skin is often complained of, and the hair becomes dry and falls out.

Objectively the patient presents a pallid or grayish skin, the eyelids are puffy, and the conjunctivæ congested. The mouth and the nose may be inflamed, and a perforation of the septum may be found. Patches of brown or blackish pigmentation may be present about the eyes, temples, or neck, and there may be a bronzing of the trunk. A skin eruption, which takes a variety of forms, may be present. The blood may show a reduction in the number of red cells.[2]

Prevention.—Protection against the breathing of arsenical fumes should be furnished the worker by the use of closed apparatus for dangerous processes and by the installation of suitable exhausts. There should be due care in the employment of raw materials free from arsenic as an impurity. Workers should be carefully instructed as to the dangers of an accidental escape of arseniuretted hydrogen and what to do in such an emergency.

The worker's body should be protected from head to foot with suitable work clothes to exclude irritating dust. Goggles should be worn and, where necessary, respirators. The face should be protected by applications of vaselin or cold cream. The worker's clothing and gloves should be frequently cleaned, and adequate facilities for bathing furnished. The worker should have periodic medical examinations and be instructed in the dangers of the trade and the need for personal cleanliness before meals and on leaving work.

In handling fluids containing dangerous quantities of arsenic, goggles, rubber gloves, and waterproof suits should be worn.

Treatment.—In acute cases, the patient should be removed from the poisoned atmosphere and oxygen inhalations given. Morphin may be needed to relieve the abdominal pains.

Chronic cases usually recover in time when removed from the source of intoxication, and efforts are made to improve the general health. A milk diet has been recommended until the gastric symptoms subside, and water in large quantity should be given.

Brass, Zinc, Etc.

Characteristic symptoms of intoxication have been known for a long time in workers exposed to fumes from brass. The exact cause of "brass founder's ague" is, however, not yet determined. It has been attributed, in whole or in part, to zinc, copper, cadmium, and to lead or arsenic which may be present, or mixtures of these metals. Good authorities believe the poisoning to be due to the inhalation of zinc oxid, and Lehmann claims to have induced a characteristic attack in a susceptible person by burning pure zinc in his presence. As neither zinc nor zinc oxid is considered toxic, he has advanced the theory that the symptoms are due to the absorption of decomposition products of cells of the respiratory tract which have been damaged by the zinc oxid. In support of this, it is known that recently destroyed lung tissue injected into the body is extremely toxic.

---

[2] Keratosis of the palms and soles is frequently present.—Editor.

About 75 per cent of brass founders suffer from intoxication, there being great difference in susceptibility. Some workers acquire an immunity.

Symptoms of Acute Poisoning.—Premonitory symptoms often appear soon after exposure to the fumes. These consist of irritation of the throat and bronchial tubes with a dry cough, headache, nausea and, sometimes, vomiting. These disturbances may disappear for a time, to return later in the day on leaving work or on reaching home. There is more severe headache, muscular pains or cramps, a more productive cough, sometimes with blood-stained sputum. Chilly feelings come on and a true "shake" develops. The teeth chatter and the patient seeks sources of heat, though neither hot drinks nor external warmth stop the feeling of cold or check the shivering. He may complain of palpitation and may suffer from asthmatic breathing. The attack may continue, with or without intermissions, from fifteen minutes to hours, and ends abruptly with profuse sweating. The patient falls asleep and awakes next day somewhat fatigued, but usually in condition for work.

During the chill the temperature may rise as high as 104° F., but it may fall to subnormal. The respirations are not affected except where asthmatic attacks occur. The pulse does not usually increase to more than 130; the blood is unchanged. The urine contains albumin and casts after the chills, but becomes normal within a day or two. Zinc may be found in the urine and the feces.

Diagnosis.—An attack of brass founder's ague resembles malaria, especially when there is fever, and may be considered such by the inexperienced victim. Such attacks of intoxication are most frequent (on account of poorer factory ventilation) in the winter months, and the symptoms of inflammation of the respiratory tract which accompany the attack may cause the physician to consider it the onset of an acute respiratory infection. The chills are, however, usually more severe, and prompt recovery upsets this theory.[3]

A knowledge of the patient's occupation is all important.

---

[3] Attacks are most frequent on Monday morning.—Editor.

Prevention.—Brass founder's ague rarely occurs where the ventilation is excellent. Aside from this means of prevention and the use of improved technical methods of work, those workers who are most immune to poisoning should be selected, and those especially sensitive advised to find other work. Alcoholism, poor food, loss of sleep, and all habits that depress vitality increase susceptibility to attacks of the "shakes." The drinking of milk before and after exposure to the fumes has been recommended.

Treatment.—During the attack a dose of magnesium sulphate or other laxative should be administered. Hot drinks (milk or lemonade) should be given, and the patient made as comfortable as possible with hot blankets and hot-water bags.

Chronic Poisoning of Workers in Brass.—From constant inhalation and swallowing of dust from the metals of which brass is composed, a considerable percentage of brass workers develop chronic disease of the respiratory and the digestive organs. Chronic bronchitis and emphysema are common, and fibrosis or tuberculosis of the lungs often develops. There is complaint of a metallic taste in the mouth; there is loss of appetite, thirst, dyspepsia, irregular bowels and sometimes colic. Nausea and vomiting may occur. Headache and muscular pains are frequent symptoms. The heart, arteries, and kidneys may show signs of degeneration. The patient suffers from mental depression and becomes prematurely aged in mind and body.

Objectively, the patient looks worn, anemic and ill-nourished or emaciated. He is short of breath and shows signs usually of disease of the respiratory organs. Arteriosclerosis is common. The teeth and gums are usually much diseased and a lead line may be present. The presence of copper may also show itself in deposits on the teeth or in green staining of the underclothes. Lead or copper may be found in the urine. The blood may show a marked reduction in red cells and hemoglobin. Basophilic degeneration may be present.

Prevention.—Shop cleanliness and good ventilation, the confining of certain processes and the use of special exhausts help in prevention; but the worker must observe due cleanliness. Respirators may be needed in certain processes. The chewing of tobacco while at work should be abandoned, as the tobacco is handled with hands covered with dust and a certain amount is taken with each chew. The worker should wash his hands thoroughly before eating, and his lunch and place of eating in the factory should be free from dust.

Treatment.—The treatment is symptomatic.

LEAD

Lead is one of the oldest known industrial poisons and still claims the largest number of victims. It is said that, for one case of industrial poisoning from other causes, there are twenty from lead. Lead is used in over a hundred industries, but the greatest danger occurs to smelters, to the makers of white lead, to workers in the ceramic industry, and to painters, especially those who work indoors.

Pathology.—Lead enters the body chiefly as dust by way of the digestive organs, though fumes and some dust are absorbed through the respiratory tract. A small amount may enter through the skin, and cases have been reported of poisoning in dentists from the rubbing of amalgam in the palms. The metal is discharged through the intestinal tract, the kidneys, the liver, in the saliva, and, to a slight extent, by way of the skin. If the output is slower than the intake, lead is deposited in the liver, kidneys, muscles, brain, in the gums (causing the appearance of the Burtonian "lead line"), and elsewhere. It may remain in the body for a long time after exposure to the poison has ceased.

Women and children are more liable to intoxication than men, and there is a great variation in individual susceptibility. The use of alcohol increases the risk of poisoning, and one attack makes the person more liable to another. Symptoms may show themselves within a few days or after more than a year of exposure.

Lead is by no means consistent in its pathological effects in different cases, but it destroys red blood-corpuscles and their hemoglobin, and in a considerable percentage of cases many of the remaining red cells show basophilic granulation. It often produces a neuritis, and, while the efferent fibers show most injury, the afferent fibers are also affected. Lesions of the brain and meninges have been noted in cases with symptoms of encephalopathy. It predisposes to arteriosclerosis and may help to bring on interstitial nephritis and gout.

Symptoms.—There is considerable variation in the clinical picture of lead poisoning. The earlier symptoms are referable to disturbances of the digestive tract, and the condition which usually brings the patient to the physician, or the physician to the patient—abdominal colic—is one of the characteristic expressions of lead intoxication. Some time before such an attack an unpleasant taste may have been noticed and there may have been loss of appetite, nausea or vomiting, constipation, loss of weight, weakness, headache, and pains in the limbs.

The attack of lead colic comes on without premonition other than that obstinate constipation may have been present for several days. The pain is located in the neighborhood of the umbilicus, is cutting in character and radiates in all directions. It seldom lasts more than a quarter of an hour but may return after a short respite. The abdomen is retracted, the muscles are rigid; pressure may increase or relieve the pain. The suffering is so intense that it may be difficult to retain the patient in bed.

Anemia is an early and, sometimes, only sign of plumbism, though it may be absent when other marked symptoms are present. There is a gradually increasing pallor which is said to be out of proportion to the blood changes. There may be destruction of red blood-cells and the number sometimes falls as low as 3,000,000, while the hemoglobin is correspondingly reduced. There may be reduction of hemoglobin with a normal red blood count.

Much has been made of the importance, for diagnosis, of basophilic granulation of red corpuscles, but Oliver found that in 70 per cent of his cases of poisoning this sign was absent, while it might be found in workmen who showed no signs of intoxication.[4]

Weakness or paralysis of certain groups of muscles, notably of those which extend the wrist, is a characteristic condition in many cases of lead poisoning. The palsy usually appears first in the extensors of the third and fourth fingers, extending to those of the other fingers and then to the muscles which extend the wrist. The hands drop when the forearms are held horizontally forward, with the palms down. Other muscles of the arms and shoulder may be affected, and sometimes the extensors of the toes, foot or leg are paralyzed. Muscles of the back, larynx, eyes and diaphragm have been affected in rare cases. Usually the muscles attacked are those which are used most in the patient's work, and the theory has been suggested that these most readily suffer because most fatigued. Atrophy and fibrillary tremors accompany the paralysis. Muscle pains and soreness may exist without signs of paralysis, and rheumatic pains in the joints are common. Paresthesia is not uncommon, and blindness from damage to the optic nerve or retina has occurred.

In a few cases, usually younger subjects, cerebral symptoms are prominent. There is severe headache and vomiting, and the patient becomes delirious or goes into convulsions. In rare instances the patient passes into coma which is fatal within a few days. These uncommon cases of "saturnine encephalopathy" more often occur in women, and, in one case, came on after only five weeks of exposure to the intoxicant.

Women workers in lead suffer frequently from menorrhagia, and "the only way for a pregnant female to reach full term is to retire from work at the earliest possible date." Children who survive after birth are said to show serious damage from lead poisoning.

In review, the subjective symptoms for which the physician is seen by the poisoned lead worker are: (*a*) those referable to digestive disturbances: bad taste, loss of appetite, dyspepsia, loss of weight, constipation, and especially attacks of colic; (*b*) those due to the effect of lead on the nervous and muscular system: pains in muscles and joints, cramps, weakness, tremor, paralysis, headache, and, more rarely, disturbances of the central nervous system; and (*c*) symptoms from the effects upon the blood—chiefly weakness.

---

[4] Leukocytosis is commonly present and nucleated reds (normoblasts) are also frequently found.—Editor.

To the examiner the signs of anemia and poor nutrition are usually the first evidence of the intoxication. The face of the patient presents a yellowish, pallid hue, and it is thin and drawn and lacks expression. On looking into the mouth, a line of discoloration varying from light slate to dark blue will often be found in the gums at the margin of the teeth—the Burtonian "lead line." This is produced by a deposit in the gum of sulphid of lead, and does not disappear on scrubbing the teeth with a brush. In many cases of poisoning it is absent, and it is often present in workers in lead who have shown no symptoms. Such a line appears also in persons who are receiving large doses of bismuth. In workers in copper a greenish blue line is often present on the teeth, though not *in* the gums. In pyorrhea there may be a grayish discoloration but not so definite a line. There may be small patches of bluish discoloration, from lead, on the inner surface of the lips or cheeks.

On asking the patient to show his teeth, a fine tremor may be seen in the muscles of the mouth. When asked to hold out his hands, he may exhibit signs of weakness or paralysis in the extensors of the fingers or of the wrists, or this symptom may be elicited by having him extend his hands against resistance. There may be weakness of other muscles and this may show in unsteadiness or peculiarity of station or gait.

The arteries may be thickened, the heart enlarged, the blood-pressure above normal and there may be signs of nephritis.

Diagnosis.—The diagnosis of plumbism may be easy when there is a history of exposure, a blue line, and attacks of constipation and colic. The presence of a wrist-drop makes it still easier.

When one is consulted in earlier stages for more general symptoms, when there is no lead line and the occupation of the patient is overlooked, mistakes as to etiology are easy. Indigestion, weakness, malnutrition, headache, insomnia and mental depression may be due to a variety of causes.

Lead colic has often been mistaken for appendicitis, but the location of the pain, the absence of fever and the history of exposure to lead should be taken into account. It has also been mistaken for gall-stone colic. There is no jaundice with lead poisoning, though the yellowish appearance of the cachectic lead worker may give some suggestion of icterus.

To distinguish a first attack of lead colic from pain due to pyloric spasm may require all the data to be had in the case.

Prevention.—Less toxic materials should be substituted for lead where practicable. In some European countries the use of lead paints has been prohibited. New processes and machines for the making of lead products which do away with, or reduce, exposure to fumes and dust should be adopted. Where fumes or dust are produced in the workroom, the ventilation should be such as to reduce these to a minimum. The walls, floors, and objects to be handled should be kept as free from dust as possible. The wearing of suitable respirators should be compulsory where ventilation is still inadequate, though these are always unsatisfactory prophylactic agents.

Rooms free from lead dust must be provided for the storing and eating of lunches. Caps and overalls of a nature to prevent the penetration of dust should be provided, and their use be made obligatory. Time should be allowed, and facilities furnished, for thorough cleansing of the face, hands, and nails with clean, running water before meals and after work. Where there is much exposure to fumes or dust, shower baths should be provided. The use of tobacco during work time should be prohibited. The taking of milk before and during work is believed to have some prophylactic effect.

The worker should be fully instructed in the dangers attendant on his trade and, in detail, as to how the lead is taken into the body. He should have frequent periodic medical examinations, and should be compelled to make use of all the means of personal cleanliness available.

Women and children should not be employed in industries involving any risk of lead poisoning.

Treatment.—For the pain of *colic* a hypodermic of atropin may be given, or tincture of belladonna. Thompson recommends the following combination:

℞ Tinct. cardamonii comp.
Aquæ menthæ piperitæ
Spir. ammoniæ aromatici āā ℥i.
M. Sig. One dram every two hours in a wine-glassful of hot water.

A hypodermic dose of morphin and atropin may be needed in severe cases. The patient should be kept in bed, and a hot-water bag or stupes applied to the abdomen. A dose of Epsom salts or of castor oil should be administered, and also an enema of warm soapsuds. Plenty of milk can be given or, if the patient is nauseated, water should be substituted.

For the *anemia,* iron may be prescribed.

For the *paralysis,* massage, electricity and efforts at reëducation are indicated.

Other treatment is symptomatic.

For the *removal of lead* from the body, the administration of potassium iodid in small doses (not more than 5 to 10 gr. daily) has been recommended, but it is of doubtful value. The use of the electric bath ("Clague-Oliver" method), which was so strongly advocated by Sir Thomas Oliver, seems to have only a mental effect. Also the benefit from sulphur baths is probably due to suggestion.

The removal of a worker from exposure to lead is advisable, save where he has reached an age when he would be unhappy in another occupation.[5]

MANGANESE

Fumes of manganese or its compounds may be absorbed through the respiratory tract, or it may be swallowed in the form of dust. Poisoning comes on after exposure for several weeks.

Pathology.—No definite pathologic changes have been discovered, but the symptoms point to injury of the central nervous system.

Symptoms.—The worker feels weak and drowsy. There is salivation, loss of appetite and weight. There may be hysterical attacks with uncontrollable laughter or crying, and sometimes more marked mental disturbances occur. There is increasing unsteadiness in standing and walking; there is difficulty in stopping in going down hill; and the victim may lose his balance and fall in stepping backward. There are rheumatic pains or cramps in the legs, and numbness of the feet. Incoördination of the hands may occur, as in writing, and in advanced cases the victim becomes helpless to care for himself.

To the physician the patient presents a masklike countenance and speaks in a low monotonous voice with slurring speech. There are involuntary muscular movements of the arms, legs, head, and trunk. The reflexes are heightened, ankle-clonus and Romberg's sign may be present. There may be a peculiar slapping gait, and, in advanced cases, propulsion and retropulsion are present. The urine, blood, and spinal fluid have been found negative.

Prevention.—Shop cleanliness and adequate ventilation would seem to be sufficient, since manganese is but slightly toxic and no cases of poisoning seem to occur where there is less than 5 per cent of it in the dust. Adequate facilities for personal cleanliness before meals and after work should be supplied. The lunch room should be free from dust.

Treatment.—Early cases recover spontaneously when removed from work, but the treatment of chronic cases is unsatisfactory. Elimination of the poison by laxatives and sweating is indicated. Rest, hydrotherapy, massage and good feeding should improve the mental symptoms.

MERCURY

Poisoning by mercury and its compounds has practically disappeared among mirror workers because of the substitution of silver nitrate in their work, but it is still common enough among hatters, instrument makers, etc. In industrial poisoning we have to do chiefly with the element or with the nitrate of mercury.

---

[5] In some patients the administration of iodids seems to increase the symptoms and they should be used with great caution. The administration of fel bovis is said to aid in the elimination of the poison.—Editor.

Pathology.—The intoxicant is absorbed through the skin or is taken in through the respiratory or digestive tracts in the form of fumes or of dust. It is eliminated chiefly by the intestine, but also in the urine, saliva, and sweat. There is great variation in individual susceptibility, and women, children, and the sickly and ill-nourished are more sensitive.

In industrial poisoning with mercury the digestive and nervous systems are most affected. Stomatitis is common. The gums swell, bleed easily, and the teeth become loosened. Yellow ulcers may develop on the cheeks or lips. Fatty degeneration of the skeletal muscles, liver, and kidneys has been reported in chronic cases. Symptoms often point to action on the central nervous system, and mercury has been found in the brain as well as in the liver. The skin is often affected.

Symptoms.—There are probably no acute cases in industrial poisoning with mercury. The earliest symptoms may be loss of appetite or slight diarrhea, weakness, and headache. The patient sometimes complains of indefinite pains in his arms and legs. The most prominent feature is soreness of the mouth, difficulty in chewing, and there may be salivation. He may notice that his hands tremble or that he has muscular twitchings elsewhere. He may, in rare cases, experience convulsive movements. Paralysis of arms or legs may sometimes occur.

A peculiar mental change may come over the worker. He becomes highly self-conscious and timid and unable to behave with his accustomed freedom in the presence of strangers. These psychic disturbances are peculiar to mercury poisoning, but are not of common occurrence. Loss of energy and apathy are more frequent accompaniments. In some cases there has been despondence and attempts at suicide.

Outwardly there are signs of impaired nutrition, and pallor may be marked. The mouth shows a copper-colored redness; the gums may be swollen or ulcerated and the teeth loose; yellow ulcers may be seen in the lips or cheeks. The breath is often fetid from the condition of the mouth. There may be an abnormal flow of saliva. Tremor of the tongue or fingers is a typical sign. Coarser and more irregular muscular movements may be present, resembling those of paralysis agitans but made worse on volition. These mercurial "shakes" have been so violent in rare cases as to hurl the victim out of bed. There is nothing distinctive in the blood, but there is usually a diminution of red cells and a corresponding or greater reduction of hemoglobin.

Of the three principal signs and symptoms of industrial poisoning with mercury, stomatitis may exist without tremor and tremor without stomatitis. Erethism is most marked in those who display the other symptoms.

Prevention.—The ventilation of rooms where workers are exposed to fumes or dust should be of the best, and this includes the regulation of temperature which should be kept at 60° F. or below, if possible. Work should be suspended when the temperature reaches 78° F. Respirators should be supplied where needed, but cannot be relied upon. The floors and furniture should be impervious to mercury and should be kept clean. Suitable work clothes should be supplied, including means of protection for the head, and these should be frequently cleaned. There should be ample facilities for bathing the hands before eating and the whole body after work, and the lockers and dressing room should be located at a suitable distance from the workroom. Potassium chlorate solution can be used as a mouth wash before eating.

Medical examinations should be periodic and frequent and the workers advised of the dangers of the occupation and the need for personal cleanliness and for avoiding contact with the poison as far as possible.

Treatment.—The patient should be removed from possibility of further poisoning, and efforts directed at restoring his general health by abundant good food and fresh air. The mouth should be cleansed frequently with a solution of chlorate of potash or of boric acid, and any ulcerations painted daily with argyrol. Recovery is usually complete in time.

NON-METALS AND THEIR COMPOUNDS

ANILIN AND OTHER AMINO COMPOUNDS OF BENZOL AND ITS HOMOLOGUES

Anilin and its near relatives, toluene, naphthalene, xylol, the naphthylamins, benzidin, toluidin, etc., have come into extensive use in connection with the rubber and dye industries and in a variety of other occupations.

Pathology.—The poisons are taken into the digestive organs as dust or through the respiratory passages as vapor; they may also produce disastrous results by absorption through the skin.

They affect the blood profoundly, chiefly by the formation of methemoglobin and hemolysis of red cells. Some authorities consider that there is also direct injury to the nervous system.

Symptoms.—The symptoms, in the order of their appearance, are: drowsy feeling, but with inability to sleep, weariness and sometimes mental uncertainty. The person becomes irritable, complains of headache, dizziness and nausea; there is loss of appetite, indigestion, constipation or diarrhea; and the patient may notice that his urine is dark or smoky. The throat becomes dry and there is a choking sensation. There are pains in the muscles and joints, and sensitiveness to cold; speech may become difficult, the memory fail, and unconsciousness ensue.

The earliest objective symptom is a flushed face which is succeeded by marked pallor with deepening cyanosis. The respirations increase; the pulse, at first rapid, becomes slow and weak; a macular eruption sometimes appears, but there is no purpura.

An examination of the blood may show a polycythemia, but this is followed by a decrease in the number of the red blood-cells, a decrease of hemoglobin, and a marked production of methemoglobin. The Tallqvist scale cannot be used because of the change in the color of the blood produced on exposure. In very acute cases the leukocytes may be increased, and some basophilic changes may take place in the red blood-cells. At least one student considers the stippling of the cells as valuable a sign as in lead poisoning.

Albumin and blood and bile pigments often appear in the urine.

Diagnosis.—The most important points in diagnosis are: a history of work in anilin, marked pallor, and cyanosis of the lips. The characteristic odor of anilin which is often present confirms the diagnosis. Such cases have been mistaken (chiefly because of the cyanosis and breathlessness) for valvular heart disease, and treated accordingly.

Prevention.—The workroom should be made safe under ordinary circumstances by forced ventilation, and the poisonous compounds should be handled in closed and ventilated receptacles. Apparatus for oxygen inhalation should be at hand. The time for working in fumes should be limited and the workman instructed as to the dangers, the early symptoms of poisoning, and the need for an immediate bath and change of clothing when the chemicals have been spilled or splashed upon him. Protective work clothing should be furnished, together with clothes rooms and bathing facilities. The hands should be protected by rubber gloves, and the feet with rubber-soled shoes. Monthly medical examinations should be made for signs of chronic poisoning, and the worker advised in matters of hygiene. Alcohol should be prohibited.

Treatment.—In mild and chronic cases the patient should be removed from work, and mild laxatives should be given. The recovery is usually complete after a few months. In severe cases artificial respiration and oxygen inhalations may be needed. Hypodermic stimulation of the heart can be tried, with digitalis or camphor. Venesection, followed by transfusion of blood, should be resorted to in the worst cases.

Benzene (Benzol), Benzin, Naphtha, and Gasoline

Benzin and benzene (benzol) are often confused on account of the similarity of name. Though of somewhat different origin, one being a petroleum and the other a coal-tar product, they are somewhat closely related; moreover, their toxic effects are similar. They have much the same uses as solvents and may be found together in the same industry, so that it may be difficult to say, in a given case, which material has caused the poisoning. Both substances often have admixtures of related hydrocarbons, such as toluene, xylene, cumol, naphtha, etc., which affect their toxicity. Industrial poisoning usually arises from such a mixture.

Serious cases of acute poisoning have occurred on exposure to concentrated vapors, as in those who have descended into storage tanks. Milder cases take place in a great variety of industries where these agents are used as solvents and for cleaning purposes.

Pathology.—Benzin and its related substances are taken into the body chiefly through the respiratory tract, but they may also be absorbed through the skin. Individual susceptibility varies greatly, and women are especially sensitive.

They have a marked action upon the blood and upon the central nervous system, the latter effect being, however, due in part to the former. There is anemia. The red corpuscles may fall to below 1,000,000 and the white cells to fewer than 1,000 per cubic millimeter. The hemoglobin content falls correspondingly, but the color index remains about normal. Hemorrhages into the mucous membranes and skin are frequent, and congestion and hemorrhage of the intestinal and urinary tracts may occur. Hemorrhages under the endothelium of the heart have been noted, and fatty degeneration of the heart, kidneys, and liver has been reported in very severe cases.

Symptoms.—Where the intoxication is not rapidly overwhelming, the worker complains of headache, dizziness, dryness of the throat, spots before the eyes, dimness of vision, and pains in the extremities. In mild cases he may exhibit a condition resembling alcoholic intoxication with mental excitement and irritability, but the stage of exhilaration is followed by one of depression, malaise and cloudy mentality. In severe cases there is mental confusion, followed by stupor or loss of consciousness with muscular twitchings or epileptiform attacks.

In chronic cases the symptoms are similar but more protracted. There is headache, dizziness, loss of appetite, tremor of the hands. There is bleeding from the gums and the nose, and Legge thinks the appearance of these signs can be used as a guide for exclusion from work, but other authorities consider this too late for safe removal. There may be bloody stools and blood in the urine. The pulse is small and weak and the respirations increase with the destruction of the blood elements. The erythrocytes and leukocytes may be greatly reduced, and the hemoglobin content falls with the destruction of red cells.

Prevention.—Adequate ventilation and shop cleanliness offer abundant protection against poisoning with these agents. Where tanks are to be cleaned or repaired, residues of the contents should be removed for a sufficient period beforehand, or the workmen should be supplied with diver's helmets. Where benzin or benzol may come in contact with the hands, the skin should be coated with some substance insoluble in the poison.

Treatment.—For acute symptoms, aside from removal from the poisoned atmosphere, fresh air and rest are needed. Artificial respiration and oxygen inhalations should be given in severe cases, and transfusion of blood should be tried when severe hemorrhages occur.

Carbon Bisulphid

Carbon bisulphid is a solvent used chiefly in the rubber industry, but cases of poisoning occur among makers of rubber cement, belt makers, shoe repairers, etc. One part in a million of air is said to be toxic and the breathing of one and a half parts per million will produce serious symptoms.

Pathology.—The poison enters the body through the respiratory organs, or by way of the skin, and it finds exit by the former channel. On reaching the blood it destroys the hemoglobin, one case showing a normal number of red and white cells but the hemoglobin reduced to 40 per cent. It acts also directly upon the nervous system.

Laudenheimer asserts that all workmen who come at all in contact with carbon bisulphid are injured by it.

Symptoms of Acute Poisoning.—Acute poisoning is rare. There is headache, dizziness, shortness of breath, palpitation, nausea, vomiting. There may be lack of coördination. Sleeplessness is a common complaint, though, instead, there may be drowsiness. The patient may have hallucinations and behave as if intoxicated with alcohol. He may lose consciousness and have convulsions. Maniacal attacks may come on without previous symptoms.

The patient looks pale, there is the odor of rotten eggs in his breath. The pupils do not react to light. The reflexes may be lost, and there may be deafness and local anesthesia.

Chronic Poisoning.—Chronic poisoning may come on after weeks or after years of exposure to carbon bisulphid. It begins to show itself in severe headaches, nausea, and dizziness. There are complaints of cold in the extremities, or of pains, cramps, numbness, or of anesthesia in different parts of the body. The vision may be affected and there may be weakness, tremor, or larger involuntary movements. The patient is mentally exalted, is abnormally good-humored and talkative, or exhibits violent fits of anger. More marked signs of psychic aberration may appear. The period of gayety and excitement is succeeded, after weeks or months, by one of as profound depression, with mental dullness, weakened memory, and difficulty in speaking. There are disorders of digestion, and there may be colic, diarrhea or constipation. The nutrition suffers in consequence, and there is anemia. The disturbances of sensation and motion of the earlier stage are increased. There may be paraplegia or hemiplegia with atrophy or contractures. The mental faculties degenerate.

Rambousek has given the following picture of the chronic case of carbon bisulphid poisoning.

> "The invalid appears in the consulting room in a bent position, leaning upon a stick, with head and hand shaking. The gait is clumsy so that the patient steps, rather than walks. When seated, the tremor ceases to some extent, but with purposive movements increases rapidly, involving the whole body, so that an exact systematic examination becomes impossible, and the invalid sinks back into the chair exhausted and bathed in perspiration. He complains of cold in his extremities; he looks pale; the skin of the upper extremities is totally without feeling, as also is the upper part of the feet; the skin of the head is hypersensitive; the muscular strength of the arms is almost lost; testing the strength brings on marked shaking followed by a fainting fit caused by exhaustion. The extremities are cyanotic; the knee jerks exaggerated. The patient suffers from indigestion, constipation, headache, and giddiness; he is irritable and depressed; his memory is weak; mental derangement cannot be proved."

Prevention.—The use of some less poisonous solvent is the ideal means of prevention, and in many factories carbon tetrachlorid or some other agent has been substituted for the bisulphid, more, however, because it is less inflammable than because it is less toxic.

Adequate ventilation is of the greatest importance. As the poisonous gas is heavier than air, the exhaust vents should be placed in the floor. Mechanical and closed methods of handling the materials should be adopted as far as possible.

Impervious work clothes, including cap, gloves, and boots, should be furnished the worker, and, as these will become impregnated with the gas, they should be washed frequently in the factory. The dressing room and the lunch room should be removed from the source of contamination. There should be adequate facilities for bathing.

Workers should be subjected to monthly examinations, and the less robust, and those showing signs of nervous instability, should be given other employment. There should be frequent change of work. In England the men in vulcanizing rooms are permitted to work only two periods of two and a half hours each per day.

Treatment.—The patient should be removed from exposure to the poison. There is no specific treatment.

Carbon Monoxid

Carbon monoxid is said to cause more accidents, fatal and non-fatal, than any other substance in industry. Aside from its presence in mixtures of gases, natural or artificial, used for furnishing light or heat, it may be present in mines and wherever there is incomplete combustion, whether in the furnace room, the tailor's shop, or the garage. One-half of one part per thousand in the atmosphere has proved poisonous and two or three parts may be dangerous to life after half an hour's exposure.

Pathology.—The effects of carbon monoxid are due chiefly to its affinity for hemoglobin and its property of supplanting oxygen in proportion to the amount of gas reaching the alveoli of the lungs. The gas is believed to have also a direct action upon the central nervous system.

Fatty degeneration of the blood-vessels and other tissues, and thrombosis and hemorrhages have been found in the brain of cases coming to autopsy. Bronchopneumonia is a frequent complication.

Acute Poisoning.—Where concentrated gas is inhaled, the victim may, without premonitory symptoms, fall unconscious, and death ensue almost immediately. In the larger number of cases, however, the worker has warning of danger in the form of throbbing or fullness of the head, roaring in the ears, blurring of vision, shortness of breath, and weakness of the legs. There may be a dryness of the throat, nausea, or vomiting, abdominal distress, pain at the base of the skull, palpitation, and a feeling of chilliness. The victim is often unable to move hand or foot, mental confusion or excitement may occur and convulsions may accompany loss of consciousness. Coma may last as long as forty-eight hours with recovery.

During consciousness the face is flushed, the pulse full and rapid, and the respirations quickened. With unconsciousness the breathing becomes shallow or intermittent, and the pulse may be full and slow, or small and irregular. The lips and conjunctivæ may be bright red, but instead there may be pallor and cyanosis. There are often patches of hyperemia on the skin.

With the recovery of consciousness, some of the early symptoms—headache, dizziness, nausea, weakness, with pain in the limbs, or paresthesia—may be present for a few days. Neuritis, paralysis, anesthesia, affections of speech, loss of memory, and mania may be persistent after effects. Pneumonia also may be a complication.

Chronic Poisoning.—Chronic poisoning from long exposure to small amounts of carbon monoxid, as in linotypists, pressers of clothes, and garage workers, results in a feeling of fatigue, headache, dizziness, dyspnea on exertion, palpitation, indigestion, loss of appetite, neuralgic pains, nervousness, sleeplessness and, sometimes, mental dulness.

The red blood-cells in such cases are often increased, in order to maintain the oxygen-carrying power of the blood, in one case to 11,200,000. The color index is usually under five-sevenths. The proportion of eosinophile cells has been found increased.

Prevention.—The prevention of carbon monoxid poisoning consists in thorough ventilation and the prevention of leaks from gas-making machines and from mains and pipe fittings. There should be adoption of the best type of furnaces, and the use of overhead, rather than underground, mains in the factory. The cleaning of flues, and other especially dangerous processes, should be conducted with due regard for the risk that may be present. A respirator which is said to be proof against 1 per cent of the gas for three hours has been devised for the U. S. Bureau of Mines.

The workers should be amply informed of any dangers of carbon monoxid exposure and of the symptoms. Competent first-aid men should always be in readiness to assist where there is possibility of poisoning. Oxygen or carbon dioxid and oxygen should be kept at hand for emergencies.

Treatment.—The treatment of acute cases consists in immediate removal of the poisoned person from the atmosphere containing the gas, and the use of artificial respiration by the Shaeffer method. Oxygen, in pure form, should be sprayed over the face through an inhalation mask. Henderson and Haggard recommend the use of a mixture of 8 to 10 per cent of carbon dioxid and oxgyen, the carbon dioxid serving to induce deeper breathing. The results of their experiments with this mixture are most encouraging. The artificial respiration should be kept up for a long time. Complications and sequelæ are to be treated symptomatically.

It is very important also that the patient should be made warm with hot blankets and hot-water bags. He should be kept flat, warm, and quiet until well recovered.

For the chronic case removal from exposure to the gas is imperative, and he should have rest, fresh air, and good food.

CARBON TETRACHLORID OR TETRACHLORMETHANE

Carbon tetrachlorid is a solvent used, on account of its greater safety, in place of benzin or carbon bisulphid.

It is closely related to chloroform and produces a similar effect on the nervous system. It is less narcotic, but more local in action. It is irritating to the eyes, nose, and throat, and also to the skin. Stronger fumes cause coughing, headache, nausea or vomiting, loss of appetite, weakness. There may be faintness or delirium and loss of consciousness.

Prevention consists in good ventilation, and *treatment* in a removal of the patient to fresh air and the use of artificial respiration if needed.

METHYL ALCOHOL

Wood alcohol is used as a solvent in the making of polishes, varnishes, and dyes, and is met with in a variety of industries.

Pathology.—Aside from its effect in producing degenerative changes in the optic nerve, no other pathological findings have been reported.

Symptoms.—When inhaled in concentrated form, it produces headache, nausea, coryza, thirst, vomiting, abdominal cramps, dizziness, chilliness, restlessness, and insomnia. There may be muscular tremor or twitchings, the pupils may be dilated and immobile, and there may be loss of vision. Blindness often comes on within twenty-four hours of exposure; unconsciousness and death may occur a day or so later.

In some cases symptoms are produced similar to those of drunkenness from the use of ethyl alcohol.

Chronic Poisoning.—Cases of poisoning of slow onset with conjunctivitis, photophobia, epiphora, amblyopia, pain, areas of paresthesia, anesthesia, and weakness have been reported.

Prevention.—The use of methyl alcohol should be abandoned, as far as possible, for some less dangerous solvent. Where used, there should be free ventilation of the workroom.

Workmen should be informed of the nature of the material used and of its dangers when employed in a closed space with insufficient ventilation.

Treatment.—The person must be removed at once from the poisoned air. In acute poisoning artificial respiration may be needed. In serious cases bleeding and transfusion may be employed.

NITROBENZOL, DINITROBENZOL AND TRINITROBENZOL

The nitrobenzols have been a source of poisoning in this country from their use in the dye industry.

Pathology.—These compounds affect the blood and blood-forming organs. They have, also, a direct action upon the nervous system, but the symptoms are believed to be due chiefly to the formation of methemoglobin.

Symptoms.—The symptoms are varied. In acute cases there is a feeling of fullness in the head with frontal headache; there is often dizziness, disturbances of vision, and mental confusion; the legs become weak and the person may be unable to stand or walk; there is nausea and vomiting. Unconsciousness may come on suddenly, though usually this does not occur until after the person has left his work.

The face is flushed or cyanotic, the lips and tongue are bluish. The temperature is subnormal, but later may run above normal.

In *chronic cases* there is headache, digestive disorder, shortness of breath, muscular weakness and twitchings, and disturbances of vision. The capacity for reading newspaper print and for writing is often lost.

The skin is pallid or grayish, but sometimes icteric. The mucous membranes of the lips and tongue are bluish. The blood is chocolate-colored from methemoglobin. The red blood-corpuscles are increased and vary in size from microcytes to megalocytes; the hemoglobin is decreased. The white corpuscles may be increased to 40,000. The liver is enlarged and tender at first, but later shrinks below normal. Ascites may follow.

Many workmen seem to become more or less tolerant to the poison, but remain anemic, cyanosed, and short of breath on exertion.

Prevention.—There should be the utmost cleanliness of the workroom, including excellent ventilation. The workrooms should not be overheated as this increases volatilization. The workmen should be instructed as to the highly dangerous nature of the compounds and as to the need for the utmost cleanliness. They should have frequent medical inspection and their physical condition kept at its best.

Treatment.—Removal from work will alone effect a cure. In acute cases artificial respiration may be needed, with inhalation of oxygen. Hypodermic stimulation of the heart may be given with caffein or camphor.

NITROGLYCERIN

The effects from nitroglycerin are experienced by the makers and users of the explosive. The poison may produce its effects when inhaled, swallowed, or absorbed by the skin. There is great variation in susceptibility. New men are especially affected. Workers seem to get accustomed to its effects while they remain at work, but the absence of a day or two renders them again sensitive. Thompson says that some persons can dip the whole arm into a pail of dynamite without experiencing any symptoms, while others can barely touch it without absorbing an amount sufficient to make themselves most uncomfortable.

Pathology.—The effects of nitroglycerin are doubtless due to its effects in producing vasodilation. There is no evidence of lasting injury.

Symptoms.—The symptoms of poisoning may come on at once, or as long as a half hour after exposure. There is a throbbing headache, beginning in the forehead and spreading. It is increased by stooping or lying. There is roaring in the ears, dizziness, disturbance or loss of vision. There may be frequent urination, and paralyses of the head, eyes, and limbs. Consciousness may be lost, the pulse becomes slow, and the breathing noisy. The face is flushed, the conjunctivæ are congested.

Those who are frequently exposed to the poison complain of restlessness, insomnia, neuralgia, disordered digestion, and tremors.

Prevention.—Proper ventilation and the use of protective gloves will reduce exposure.

Treatment.—Quiet, rest, and fresh air usually produce a cure in three or four days.

Phosphorus and Phosphorated Hydrogen

The substitution of red phosphorus and the sesquisulphid for white or yellow phosphorus in the making of matches has practically done away with that fearful industrial disease—"phossy jaw," which was characterized chiefly by a periostitis and necrosis of the mandible or maxilla. Poisoning still occurs, however, from phosphorated hydrogen or phosphin which is liberated in the making of the sesquisulphid, in the preparation of acetylene, along with arseniuretted hydrogen in the making of ferrosilicon and in some industries in which phosphorus may be present as an impurity of the materials used.

Pathology.—Phosphorated hydrogen is a dangerous poison. It produces inflammation of the lungs and damages the central nervous system. It also unites with the hemoglobin of the blood.

Symptoms.—The symptoms are difficulty of breathing, burning in the chest, cough, headache, roaring in the ears, dizziness, weakness, nausea, and, in severe cases, loss of consciousness and death.

Prevention.—Good ventilation with special exhausts should be installed where the poison is present. The employees should be fully informed of any possibility of poisoning.

Treatment.—The patient should be removed at once to pure air by trained first-aid workers, and artificial respiration given if needed. After effects should be treated symptomatically.

Sulphurated Hydrogen

Sulphurated hydrogen is met with in mines, in sewers, and is developed in manufacturing processes of a number of industries. It is often associated with carburated hydrogen, carbon bisulphid, and other gases. The breathing of an atmosphere containing one part per thousand may prove fatal in a short time.

Pathology.—The gas has a local irritant action on the respiratory organs, and, when absorbed in quantity, depresses the activity of the brain and paralyzes the respiratory center. It also reacts with the alkalis of the blood, forming an alkaline sulphid.

Symptoms.—Inhaled in concentrated form, sulphurated hydrogen produces almost immediate loss of consciousness, and the respirations cease in a few seconds.

In smaller quantity, there is loss of consciousness lasting several hours.

which is followed by profounder coma, convulsions, and death, or there may be recovery. The onset of fatal symptoms may be delayed for as long as a half hour.

The symptoms of exposure to dilute vapor are irritation of the eyes, nose, and throat, with lacrimation, sneezing, coughing, soreness of the mouth and increase in flow of saliva. Headache, dizziness, weakness, and mental dullness, nausea and vomiting come on some time after exposure, and the patient becomes anemic and loses flesh.

Prevention.—Precautions should be taken on entering mines or sewers to test the quality of the air by means of pigeons or a candle, and by efforts at forced ventilation. In factories adequate ventilation should be provided. Where danger is suspected, an oxygen helmet should be worn, and arrangements made for the safe removal of the workmen at sign of danger. Provision for resuscitation and trained first-aid people should be provided when prevention is known to be uncertain.

Treatment.—The patient should be removed at once from exposure to the poison, and, if the respiration shows signs of failure, artificial breathing should be begun at once. Blood transfusion should be tried in serious cases.

Tetrachlorethane or Tetralin

Tetrachlorethane is used as a solvent instead of turpentine in making airplane varnishes, and in the manufacture of artificial pearls, artificial silk, films, etc.

Pathology.—Tetrachlorethane is absorbed through the respiratory tract and probably through the skin. It influences the blood-forming tissues and affects the liver especially, producing an inflammation of the smaller bile ducts, fatty degeneration of the liver cells with "toxic jaundice" and, in severe cases, necrosis of these cells. There may also be fatty degeneration of the kidney.

Symptoms.—The earliest symptoms of poisoning are: abnormal fatigue, profuse sweating on slight exertion, nervousness or restlessness and discontent, inability to concentrate the attention, insomnia, loss of appetite, constipation, and consequent loss of weight. In severe poisoning there is nausea, vomiting, and sometimes diarrhea, and generalized abdominal pains. In the most serious cases jaundice appears, usually on the third or fourth day; the patient becomes unconscious and may die.

Objectively, there is, in the more serious cases, jaundice, from a slight discoloring of the conjunctiva to a yellowing of the skin of the face and chest. In severe cases the liver is enlarged and tender, and bile may be found in the urine. The blood shows an increase in large mononuclear cells, the number often reaching 40 per cent. An increase above 12 per cent is considered the first sign of reaction to the poison and a signal for close observation of the worker; immature mononuclear cells may appear, there is a slight increase in the total number of white cells, and there is a slight but progressive anemia.

Prevention.—Some safer solvent should be substituted. In France and Great Britain the use of tetrachlorethane has been abandoned in the making of airplanes.

The worker should be protected by the inclosing of hazardous processes and adequate exhausts with outlets in the floor on account of the heaviness of the fumes. Masks for emergencies should be at hand. The temperature of the workroom should be kept from rising unduly to prevent volatilization. Special clothing should be furnished, with facilities for dressing and for bathing. The dressing rooms, bathrooms and lunch rooms should be located at a safe distance from any fumes. There should be short hours and frequent vacations.

The foremen should be instructed in the symptoms of poisoning, and should relieve any worker who appears ill. The employees should be inspected every two or three days by the physician, who should examine the blood of any suspicious cases. Where the change in mononuclear cells indicates poisoning, the worker should be excluded from work for at least a week and should be carefully watched. In cases of any severity, the man should not return to work, and two mild attacks should also be a sign for rejection.

Trinitrotoluene (TNT)

Trinitrotoluene (TNT, Trotyl) is used chiefly in the making of explosives. It finds entrance to the body in the form of dust or vapor, through the skin, but may also be absorbed by way of the respiratory or digestive tracts. There is much variation in susceptibility to the poison, but no tolerance is acquired.

Pathology.—The blood-forming organs are attacked and, in severe cases, there is "toxic jaundice" with degenerative changes in the liver resembling those of acute yellow atrophy. In a few cases the organic changes have developed many months after absorption of the poison.

Symptoms.—The earlier symptoms are referable to changes produced in the blood. There is shortness of breath on exertion and dizziness; headache may be prominent, and an overpowering drowsiness and feeling of fatigue and cramps in the muscles. There is loss of appetite, a bad taste, nausea and vomiting in the morning, and constipation. Epigastric pain may be a significant and, sometimes, only complaint. There is often, where the worker is exposed to dust, irritation of the conjunctiva, nose and throat, and there may be nosebleed. Eczematous eruptions of the skin are common, with severe itching. The urine may be brownish in color.

When seen by the physician, the patient looks weary and dull. When not stained yellow by the poison, the face is pale or ashen, the lips bluish, and the scleræ may be yellow. The pulse is weak and the respirations quickened. There may be a rash on the hands and the face. There may be some swelling of the extremities, and purpuric spots may appear. A palpable, tender liver is a serious sign.

Blood drawn into a tube often appears a vivid yellow at the top of the clot. There is a primary anemia with a rapid fall in the number of red corpuscles, the appearance of damaged and immature cells and decrease in hemoglobin. The white cells may be increased or reduced in number. The symptoms usually run parallel with the abnormalities in the blood picture, but there may be marked symptoms with little alteration in the blood.

The Webster test is considered a guide to the amount of TNT being excreted by the kidneys, but, as the test may be positive with no symptoms, it is not a practical aid in determining poisoning.

Prevention.—The workmen should be protected by shop cleanliness and by exhausts from dust and fumes, and machine processes should be used as far as possible to prevent exposure. High temperatures and humidity should be avoided as far as possible. A complete set of work clothes with elastic bands at the wrist and ample covering for the ankles should be provided and cleansed when soiled. There should be ample facilities for personal cleanliness and for escaping all contact with the poison during meals.

There should be medical inspection, with examination of the blood of all exposed workers every week, and the foremen and all workers should be instructed as to the dangers of the occupation, the nature of the symptoms of poisoning, and the need for cleanliness. Soap and water do not remove all the poison from the skin, but a 10 per cent solution of sodium sulphite in water is said to be effective.

Treatment.—In case of the appearance of signs or symptoms, the work should be abandoned, and fresh air, laxatives, plenty of water and good food prescribed. If jaundice is present, the patient should be given a hot bath and be put to bed, and a purge and an enema be administered. Warm milk in increasing amounts up to 4 pints a day is believed to do good in such cases. Wilcox recommends the administration of about 30 gr. each of sodium bicarbonate and sodium citrate every two hours, and small enemas of sodium carbonate and dextrose every eight hours.

TURPENTINE

Turpentine is probably responsible for much more ill health than many substances that have a worse reputation.

Pathology.—Turpentine is taken into the body chiefly through the respiratory passages and it is eliminated by the kidneys. It acts as a local irritant and, when it reaches the blood, is said to have an exciting effect upon the central nervous system. When absorbed in sufficient quantity it causes inflammation of the urinary tract.

Symptoms.—Besides its irritant action upon the conjunctivæ and skin, turpentine may produce rhinitis, hoarseness, cough, headache, ringing in the ears, and dizziness, and the worker may become depressed and irritable.

In acute cases there may be nausea and vomiting and diarrhea, with frequent urination and the passage of blood, albumin, and casts. The urine has the odor of violets.

The patient becomes anemic and emaciated. Evidence of nephritis was found in 14 out of 62 turpentine workers examined by Nicholl, Flinn, and Hayhurst. Basophilia may appear.

Prevention.—There should be good ventilation when turpentine is handled, especially in confined spaces.

Treatment.—Mild cases need only pure air. Nephritis should be treated with rest, warmth, and water. Other symptoms should be treated in the usual manner.

MISCELLANEOUS

A number of materials besides those already described are sometimes met with as sources of intoxication in industry.

Some of these produce their effects chiefly from their irritative or destructive action upon the respiratory tract. The fumes of *Sulphuric Acid, Sulphur Dioxid, Hydrochloric Acid, Nitric Acid, Fluoric* and *Hydrofluoric Acids* have such an effect, as have also *Diethylsulphate* and *Dimethylsulphate, Acridin, Acrolein,* and *Acetaldehyd. Chlorin, Bromin, Ammonia,* and *Phosgine* have somewhat similar action.

Some *Chromium* compounds, besides producing a dermatitis, cause perforation of the nasal septum, and occasionally produce constitutional effects, including nephritis.

Poisoning from *Cyanogen* or its compounds in industry is rare, but may occur. When inhaled in concentrated form, it produces its well-known effects from internal administration. The person falls suddenly, breathes with difficulty, the pulse soon becomes feeble, and coma and death result.

Chronic poisoning of those handling *Prussic Acid* and the *Cyanids* has been described. There is oppression in the chest, difficulty in breathing, palpitation, dulness, exhaustion, nausea, vomiting, weakness, culminating in death. Saline injections, bleeding, and the administration of sodium thiosulphate solution are recommended.

In the chemical industry poisoning with *Iodin* and with *Methyl Bromid* has occurred, both of them producing acute symptoms referable to the nervous system.

Symptoms of *Nicotin* poisoning have been found in female workers in tobacco during their first six months of employment. Older employees do not display the same disturbances, and the ailments to which they are subject may be as much due to dust and to sedentary work as to any specific poison.

Cadmium is said to produce digestive disturbances, weakness, mental dulness and coma, and, as it is present in zinc ore, is said to be a cause of poisoning in zinc spelters. Similar, though milder, symptoms have been attributed to *Copper,* and true copper poisoning is said to have been produced in animals.

Attention has been called lately to symptoms of intoxication which are due to exposure to fumes and dust from *Tellurium* in lead and silver refineries. It is said to act characteristically in checking the flow of sweat, saliva, and gastric juice. There is dryness of the skin and mouth, gastrointestinal disturbances, and drowsiness. The prevention of poisoning from this substance is to be found chiefly in good ventilation. The treatment is symptomatic.

SOME OF THE IMPORTANT INDUSTRIAL POISONS WITH THEIR MORE CHARACTERISTIC EFFECTS

Poison	*Characteristic Effects*
Anilin and its analogues	Hemolysis and formation of methemeglobin.
Antimony	Eosinophilia and leukopenia.
Arsenic	Destruction of red corpuscles.
Benzene and Benzin	Marked reduction in red and white blood-cells.
Brass	"Brass founder's ague."
Carbon bisulphid	Destruction of hemoglobin, mental excitement followed by mental depression.
Carbon monoxid	Replacement of oxygen in oxyhemoglobin by carbon monoxid, with asphyxiation.
Carbon tetrachlorid	Similar to chloroform.
Lead	Anemia, constipation, colic, weakness, or paralysis of extensor muscles, muscle pains and cramps.
Manganese	Hysteria, incoördination, propulsion, retropulsion.
Mercury	Tremor, stomatitis, erethism.
Methyl alcohol	Blindness.
Nitrobenzols	Formation of methemoglobin, increase in number of red and white blood-cells.
Nitroglycerin	Dilation of peripheral blood-vessels.
Phosphorus	Osteitis of mandible and maxilla.

Tetrachlorethane	Increase in large mononuclears, and toxic jaundice.
Trinitrotoluene	Toxic jaundice.
Acetaldehyd	Primary effect an irritative or destructive action on the respiratory tract.
Acridin	
Acrolein	
Ammonia	
Chlorin	
Diethylsulphate	
Hydrochloric acid	
Hydrofluoric acid	
Methyl sulphate	
Nitric acid	
Sulphurated hydrogen	
Sulphur acid	

REFERENCES

Extensive treatment of the Industrial Poisons will be found in the following books, which contain numerous references to older literature:

Kober and Hanson. Diseases of Occupation, P. Blakiston's Son & Co., Philadelphia, 1916.

Oliver, Thomas. Dangerous Trades, J. Murray, London, 1902.

——— Diseases of Occupation, E. P. Dutton & Co., New York, 1908.

Rambousek, J. Industrial Poisoning, translated and edited by Legge, E. Arnold, London, 1913.

Thompson, W. G. Occupational Diseases, D. Appleton & Co., New York, 1914.

The U. S. Bureau of Labor Statistics has issued some excellent monographs, including bulletins on:

Carbon-monoxid Poisoning.
Lead Poisoning.
Poisoning in the Making of Coal-tar Dyes.
Poisons used in the Making of Explosives.
Poisons used in the Rubber Industry.

Excellent articles on Industrial Poisoning are appearing in the Journal of Industrial Hygiene, Boston, which published its first issue in 1919.

CHAPTER XXXV

OCCUPATIONAL DISEASES DUE TO PHYSICAL AGENCIES

WADE WRIGHT AND JOHN W. S. BRADY

COMPRESSED AIR ILLNESS, OR CAISSON-DISEASE

Compressed air illness, or caisson-disease (divers' palsy, "the bends," "courbatures"), is a condition produced in individuals who, having breathed air under pressures considerably beyond that of a single atmosphere, usually more than two, are subjected to an excessively rapid reduction of air pressure.

Diving naked has long been practiced, particularly in search of pearls. The diving bell, forerunner of the modern caisson, was used about the sixteenth century, but caisson work as a phase of engineering practice dates its development from 1839, when Triger, a French engineer, employed a caisson in sinking a shaft near Chalons in connection with coal-mining operations. Cochrane, an Englishman, had taken out a patent on a caisson shortly before, in 1830.

During the past few decades the construction of tunnels, bridges and large buildings has demanded the employment of many workers under compressed air. Though compressed air illness was described by Triger in 1841 and by Pol and Watelle in 1854, though but little later the probable cause of the ailment was determined by Hoppe-Seyler, 1857, it is only within the last few years that there has been evidenced any determination to avoid the needless occurrence of large numbers of cases of the disease in connection with every sizable engineering undertaking making use of caisson workers.

As in a glass tumbler, inverted and plunged into water, the retained air prevents the water from filling the tumbler, so does the compressed air in a caisson, lowered to a river bottom where a bridge pier is to be constructed, prevent the flow of water into the caisson. Analogous conditions are encountered, of course, if the caisson is thrust into soft, water-bearing soil. A subaqueous tunnel may be considered as but a lateral extension of a caisson.

To prevent the ingress of water, air must be supplied within a caisson at a pressure dependent upon the depth of the working level below the surface of the water in which the caisson is sunk. A caisson worker or diver employed 33 feet below the surface of a stream, for example, would be obliged to work in air under a pressure of two atmospheres or 15 pounds in excess of normal atmospheric pressure.

It is obvious that a worker entering a caisson in which the air is held at an absolute pressure of 30 pounds cannot pass directly from air under 15 pounds pressure. Caissons are, accordingly, supplied with chambers known as air locks. The lock is essentially a compartment of heavy steel plate equipped with two doors opening inward, or toward the working face, a pipe for the delivery of compressed air into the compartment and a valve for the release of compressed air. The customary equipment is much more elaborate, including lights, perhaps a telephone and gages for the registering of air pressure.

The worker about to enter the caisson takes his place in the air lock, the outer door is closed upon him and the air pressure within the lock elevated to that maintained in the caisson itself. The worker may then open the inner door and pass into the caisson. This process of "compression" may be accomplished rapidly with safety even when the pressure attained is relatively high. It is customary to effect compression to a gage pressure [1] of from 30 to 40 pounds in several minutes.

When the passage of the worker is from the caisson toward the outer air the procedure is reversed. He takes his place in the air lock under the pressure prevailing in the caisson, closes the inner door and permits air to be released through an exhaust valve until an absolute pressure of one atmosphere obtains. It is in this process of "decompression" that the danger of compressed air work lies and it is at this time or soon after that the symptoms of compressed air illness may develop.

In deep-water diving, there is presented a situation closely analogous to caisson work. The diver equipped with diving dress undergoes compression as he descends into the water. Air is supplied through a flexible tube at a pressure equal to or slightly in excess of the pressure of water at the outlet valve of the diver's metal helmet. Decompression is effected as the diver ascends toward the surface.

Hoppe-Seyler offered in 1857 an explanation of the varied manifestations of caisson-disease, and Paul Bert in 1878 demonstrated experimentally the validity of Hoppe-Seyler's theory. This theory was that the disease was brought about by the presence in body tissues of gas bubbles liberated during the period of decompression of individuals subjected to abnormally high air pressures. Other hypotheses regarding the origin of compressed air illness have been advanced, for the most part explaining observed phenomena either as the results of mechanical congestion, air pressure supposedly driving the blood of the peripheral circulation into deep tissues; or as the effects of the accumulated products of a metabolism whipped to increased activity by an increased oxygen supply. It is now, however, an accepted belief that the liberation of nitrogen bubbles in body tissues causes the disease.

---

[1] Gage pressure is that pressure in excess of normal atmospheric pressure.

Inhaled gases are dissolved in the blood of the pulmonary circulation in concentrations proportionate to the partial pressures of such gases in the inhaled air, a phenomenon expressed in Dalton's law.

A caisson worker employed under two atmospheres, or a gage pressure of 15 pounds, receives with each volume of inspired air twice the amount of air and so twice the amount of oxygen, nitrogen and other air constituents which he would receive were he working under normal atmospheric pressure. His blood is almost immediately saturated with the inhaled gases, but as blood gases must be conveyed throughout the body, and, because of variations in the gas absorption of different tissues, complete saturation of the body is but slowly accomplished.

All body tissues have not the same capacity for holding gases. Fat, for example, has been found by Vernon to take up about five times as much nitrogen as blood absorbs. If the body be about one-fifth adipose tissue, it is essential that, in estimating the relation of the blood weight to body weight, allowance be made for the high saturation point of fat. Reckoning the blood as of one-twentieth the total body weight, Van Schrötter considered that in reference to nitrogen the blood should be considered as of about one thirty-fifth of the body weight. It has been stated by Haldane that a body becomes half saturated in about twenty-five minutes and approximately wholly saturated in about ninety minutes. Practical experience with caisson workers and caisson-disease led Keays to believe that saturation was not usually complete until after three hours at work.

When a workman who has been under abnormal pressure is subjected to decompression, the gases dissolved in body fluids and tissues tend to assume the same relative pressure within the body and without. Gases pass from the blood to alveolar air and from body tissues to the circulating blood. If the saturation has been fairly high and the process of decompression effected rapidly, gas bubbles are formed in the tissues and in body fluids.

The gases existing in such bubbles have been found by Bert and others to be about 80 per cent nitrogen, with 15 to 20 per cent carbon dioxid and a trace of oxygen. Leonard Hill reported that in his experiments from 80 to 87 per cent of the gas set free was nitrogen, accounting for the presence of carbon dioxid and oxygen in stating his belief that "when the nitrogen is set free as bubbles, these act as a vacuum for carbon dioxid and oxygen and some of these gases are set free out of chemical combination with the blood. The oxygen is mostly used up by the tissues of the dying animal and thus we find the chief gas besides nitrogen is carbon dioxid."

The construction of a subaqueous tunnel entails usually the sinking of a shaft relatively near the margin of the water under which the tunnel is to be driven. The shaft may be an open excavation or sunk with a caisson. At the bottom of the shaft a "shield" is assembled, this being a sort of gigantic biscuit cutter which is thrust forward by a series of hydraulic jacks bearing upon the rim of the shield and, behind, upon the firmly set tunnel lining of concrete and of wooden or metal forms.

Beyond the air-tight bulkhead which walls off the tunnel from the shaft, air is delivered from a reservoir fed by air compressors housed in a building on ground level. Exhausts provide for a circulation of air and for the maintenance of proper pressure. Valves in the air line prevent the back flow of air from the tunnel. Built into the bulkhead are usually three locks, one for the passage of men to and from the tunnel, a "muck lock" for the transportation of supplies into the tunnel and the removal of excavated material, and the third for emergency use.

On "locking in," as the air pressure is gradually increased, the worker may experience discomfort in the ears due to the inequality of the air pressure in the lock and that within the middle ears, a sensation readily relieved by swallowing or by expelling the breath against the closed mouth and nostrils. There is at this time danger of rupture of the tympanic membrane. Middle-ear infections and resultant mastoiditis are not manifestations of caisson-disease, but they are conditions frequently related to work under compressed air.

As compression goes on, the worker notes a sense of increasing warmth and may begin to perspire freely. An individual unaccustomed to increased air pressures notes with astonishment the freedom of motion which is possible under even a moderate gage pressure and finds his arms swinging high and his stride prodigious as he enters the tunnel from the lock. Though such gross motor functions are favored, whistling is difficult or impossible of achievement and the voice sounds thin and high-pitched. The blood-pressure is unaffected and the rates of pulse and respiration do not alter notably.

When undergoing decompression, as the air pressure within the lock falls, the temperature of the chamber drops markedly and it may be essential that the worker be provided with heavy outer clothing or that the lock be heated. There is relatively little discomfort in the ears at this time.

Caisson-disease does not affect workmen while they are in compressed air under constant pressure. The onset of symptoms occurs following decompression and very rarely during decompression.

Of the 3,692 cases occurring in connection with the construction of the Pennsylvania East River tunnels, New York City, 1906 to 1909, reported by Keays, Erdman and others, 50 per cent developed within 30 minutes after decompression, 85 per cent within 1 hour, 95 per cent within 3 hours and 1 per cent after 6 hours, the latter group including 4 isolated and somewhat doubtful cases reported to have occurred between 15 and 23 hours after decompression.

Of 436 cases occurring on work done under the supervision of the Public Service Commission of New York, reported by Levy, 64.2 per cent occurred within 1 hour after decompression, 88.8 per cent within 3 hours and 7.6 per cent between 4 and 18 hours.

Levy states that during the construction of the Public Service Commission tunnels 680 cases, or 0.05 per cent, of caisson-disease were reported out of 1,361,461 decompressions and that in the Pennsylvania tunnels 3,692 cases, or 0.66 per cent, were reported out of 557,000 decompressions.

The mortality from caisson-disease has shown on the whole a very distinct reduction coincident with increasingly efficient administrative control of compressed air work. The following table combines data published by Erdman and by Levy.

REDUCTION OF MORTALITY FROM CAISSON-DISEASE IN RECENT YEARS

Date	Work	Maximum Pressure	Cases	Deaths	Percentage
1839	Douchy mines	37.5	63	2	3.2
1870	St. Louis bridge	50.0	129	14	10.8
1873	Brooklyn bridge	34.0	110	3	2.72
1874-1907	Hudson tunnel	42.0	1,573	3	0.19
1895	Danube bridge	37.5	326	2	0.625
1909	East River (Pa.) tunnel.	42.0	3,692	20	0.54
1921	Public Service Commission	47.5	680	2	0.29

In the following table mortality is based upon the number of decompressions.

MORTALITY BASED ON NUMBER OF DECOMPRESSIONS

Work	Decompression	Deaths	Percentage
Admiralty Commission Report	60,000	4	0.0066
Pennsylvania tunnels	557,000	20	0.0035
Public Service Commission tunnels....	1,361,461	2	0.0001

The most important symptoms of caisson-disease are pain in one or more extremity, pain in the abdomen, cerebrospinal disorders, vertigo, dyspnea and prostration or collapse. There have been observed, in addition, eye disorders, such as temporary blindness, nystagmus and diplopia; itching and hemorrhagic mottling of the skin and other minor symptoms associated with more important evidences of disease. Levy presents the following table comparing the proportionate incidence of various symptoms.

Proportionate Incidence of Various Symptoms

Condition	Per cent in Pennsylvania Tunnels	Per cent in Public Service Commission Tunnels
Localized pain	90.30	91.7
Central nervous system disorder	2.16	1.6
Vertigo	5.33	6.5
Chokes	1.62	0.1
Partial or complete unconsciousness	0.46	0.1

Localized pain in the extremities or the abdomen, a condition popularly known among compressed air workers as "the bends," was found to be the only symptom in over 90 per cent of the cases reported by Keays and Levy. The pain is usually in one joint, most often a knee. According to Keays the pain is in many instances slight and of short duration, but may be severe and last for several days. It may be localized or radiating, constant or intermittent, lancinating or a dull ache. He believes the pain to be due to local irritation, possibly to gas bubbles beneath fascia or within bursæ or tendon sheaths. Hill suggests the presence of emboli in bone marrow as a cause of pain in the extremities.

Subcutaneous emphysema has been observed and is described by Keays and Erdman. Mottling of the skin, attributable to capillary embolism, has accompanied many severe cases of prostration and is regarded as indicative of an exceptionally high free nitrogen content of the blood. Persistent itching not uncommonly follows decompression, due likely to gas bubbles in the subcutaneous tissues.

Abdominal pain was noted in about 5 per cent of Levy's series. Keays attaches great importance to abdominal pain for, while in a large number of cases it was not related to severe illness, it was in others the earliest evidence of disease which later proved to be very serious. It is often associated with lesions of the spinal cord. Nausea and vomiting have been noted occasionally, particularly in connection with vertigo and with severe disturbance of the central nervous system.

A relatively small proportion of cases have shown symptoms such as monoplegia or diplegia, referable to cortical irritation. Sensory and motor disorders due to cord lesions are more common, manifested usually by pain or numbness in one or both legs, abnormal reflexes, retention of urine or urinary and fecal incontinence. A transverse myelitis develops not infrequently among severe cases.

Vertigo, or "the staggers," is fairly commonly encountered among compressed air workers, alone or associated with other symptoms of caisson-disease. Snell noted deafness in connection with many cases of vertigo and ascribed the condition to embolism in the labyrinth. It is possible that cerebral irritation is accountable for certain cases of vertigo.

Dyspnea, often accompanied by a sense of constriction of the chest, is so well known to compressed air workers as to be familiarly designated "the chokes." Keays notes the similarity of the condition to bronchial asthma. Certain men he observed, who were subject to repeated attacks, had some degree of emphysema and following attacks would for several days present evidence of bronchitis.

A small percentage of reported cases showed partial or complete unconsciousness with collapse. Of these, many first had pain in the extremities or abdomen or displayed other evidences of caisson-disease. In an important proportion of the cases, however, collapse was sudden. Of Keay's 17 cases of collapse, 9 were fatal, although most were subjected to recompression.

Once the effects of caisson-disease have become established, there is little if any tendency for them to become increasingly severe, but, on the contrary, most affected individuals tend to recover. Complications, secondary to the disease itself, may lead to death. Transverse myelitis may result in extensive bed sores and progressive exhaustion ultimately fatal. Cases in which urinary retention exists frequently develop cystitis and an ascending urinary tract infection. There have been reported cases of long-continued and probably permanent disability resulting from myelitis (Keays and Bassoe).

Postmortem examinations of the bodies of experimental animals or men dying of compressed air illness have shown in many instances a wide distribution of gas bubbles in body fluids and tissues, in the cardiovascular system, especially in the veins, in the right side of the heart and in the vessels of the pulmonary circulation. The left heart may be found wholly emptied, the right filled with bloody froth.

Levy observed in the 2 fatal cases which he reported gas bubbles in the submucous layer of the small intestines having "the appearance of small grains of tapioca packed closely together in patches."

Boycott and Damant, studying the distribution of gas bubbles in the spinal cords of goats affected by decompression, concluded that bubbles were much more abundant in the white matter than in the gray matter (the white matter contains more fat than the gray) and that the dorsal and upper lumbar segments contained more bubbles than those of the cervical and lumbar enlargements, the former being portions of the cord less well supplied with blood-vessels. Persistent paralysis was apparently due in all cases to a necrosis of the white matter in its central parts rather than toward the periphery of the cord, an effect produced by gas emboli blocking circulation.

PREDISPOSING FACTORS IN CAISSON-DISEASE

Experience gained in the laboratory, in caisson and tunnel work, and with deep-water divers clearly indicates that the etiology of caisson-disease is determined essentially by: (1) the degree of pressure to which an individual is subjected; (2) the time under pressure; and (3) the duration and method of decompression.

There is little evidence of caisson-disease resulting after work in pressures up to 15 pounds. Levy reported that no case occurred in the East River tunnels until a pressure of 15 pounds was attained, although 188,496 decompressions had taken place. One case occurred at a pressure of 15 pounds. Up to 22 pounds pressure, in men working 8 hours with a half-hour interval at normal pressure, out of 621,342 decompressions 16 slight cases developed. Authentic reports of fatal cases occurring after exposure to pressures of less than 28 pounds are not to be found. One death, reported by Keays, resulted after 4 hours' work at 28 pounds. In the East River tunnels the lowest pressure from which decompression proved fatal was 34 pounds. It is unlikely that there is notable danger in very rapid or even instantaneous decompression from pressures up to one atmosphere.

Japp has expressed the belief that it is safe working procedure to decompress in such a manner and in such time as to have a maximum air saturation of the body on emergence from the air lock equivalent to a pressure of 25 pounds.

Closely related, however, to the matter of the degree of pressure under which work is done is the question of working shifts. Keays concluded from an analysis of the cases of caisson-disease developing in the Pennsylvania tunnels that saturation of body fluids and tissues is practically complete at the end of 3 hours. Comparing the numbers of cases occurring after 6-hour shifts (two 3-hour periods with a 3-hour intermission) and after 8-hour shifts (two 4-hour periods with a half-hour intermission) at pressures slightly in excess of 30 pounds, he was of the opinion that the shorter shift presented no great advantage, that saturation was not furthered to any important degree by the extra hour of work in each period and that consequently the dangers of decompression were practically as great after the shorter work period as after the longer. He further believed that there was additional hazard in dividing the working shift into two periods, thus doubling the number of decompressions, that with pressures from 32 to 34 pounds a continuous shift of 6 hours was preferable to two periods of 3 hours each, and that using 8-hour shifts instead of 6-hour shifts would require the employment of fewer men and so reduce the accident risk.

Levy reports that in the Public Service Commission tunnels under the East River, when men worked for 2 hours (two 1-hour periods with a 4-hour interval) at pressures ranging from 41 to 45 pounds, the percentage of cases, based on the number of decompressions, scarcely exceeded 0.1 per cent, while in the Pennsylvania tunnels during a single month when men worked at 40 pounds for two 3-hour periods (with an interval of 3 hours) there was a caisson-disease incidence of 1.63 per cent.

In Levy's series the incidence peak was reached under a pressure of 29 pounds (0.24 per cent), with a secondary peak under 34 pounds (0.15 per cent). The incidence rates at 30 pounds and 35 pounds were, respectively, 0.04 per cent and 0.07 per cent.[2]

From 22 to 30 pounds pressure the men worked two 3-hour periods with an interval of 3 hours. From 30 to 35 pounds they worked two 2-hour periods with an interval of 2 hours. From 35 to 40 pounds they worked two 1½-hour periods with an interval of 3 hours. It is Levy's belief that the incidence under 29 pounds pressure could have been reduced greatly by introducing the shorter shift at that pressure instead of at 30 pounds, and that, similarly, the incidence under 34 pounds could have been lowered by introducing a shorter shift at that pressure rather than at 35 pounds.

During recent years there has been almost universal acceptance of the principle of varying the length of working periods inversely with the pressure.

It should be noted that in most of the data relating to length of shifts there are considered only the hours of labor at the working face or other place of employment within the caisson, no allowance being made for time spent within the caisson in passage to such place of work from the outer air lock, and in return.

It is a matter of important bearing upon the incidence of caisson-disease and upon regulations governing hours of labor.

The New York State regulations effective March 1, 1920, in discussing hours of labor of men employed "in any compartment, caisson, tunnel or place" under compressed air at specified pressures, uses the words "to work or remain therein" in setting forth the permissible duration of working periods. In the rules effective May 1, 1922, are used the words "the working time . . . shall be. . . ." Provision is made for the hours of labor according to the following table:

SHIFTS AND INTERVALS OF WORK FOR EACH 24-HOUR PERIOD

Pressure in Pounds	Hours			
	Maximum total	Maximum First Shift in Compressed Air	Minimum Rest Interval in Open Air	Maximum Second Shift in Compressed Air
Greater than normal and up to 21.	8	4	½	4
Greater than 21 and up to 30....	6	3	1	3
Greater than 30 and up to 35....	4	2	2	2
Greater than 35 and up to 40....	3	1½	3	1½
Greater than 40 and up to 45....	2	1	4	1
Greater than 45 and up to 50....	1½	¾	5	¾

---

[2] Calculated upon data published by Levy.

The degree of pressure in great measure determines the length of shift; the degree of pressure and the duration of exposure to that pressure determine the rapidity of decompression.

Though the essential nature and causes of caisson-disease are recognized, there are predisposing and contributory factors which warrant consideration.

Ventilation.—In 1896 Snell endeavored to explain caisson-disease as due to carbon dioxid and, though his theory has been found unsound, much attention has been given to the carbon dioxid content of air in caissons, tunnels and diving dresses. Haldane and Priestly have demonstrated the regulatory response of the respiratory apparatus when an animal is subjected to abnormally high concentrations of carbon dioxid in inspired air. It is likely that a pressure of 2 per cent of an atmosphere of carbon dioxid would be little noticed by a workman. Hill states that as a working rule carbon dioxid should be kept down to a maximum of about 1½ per cent of an atmosphere. (This refers to the partial pressure, 1.5 per cent of an atmosphere at 30 pounds gage pressure being equivalent to 0.5 per cent of an atmosphere at normal pressure.)

McWhorter stated that the maximum carbon dioxid pressure attained in the Pennsylvania tunnels was 0.1 per cent, or 0.3 per cent at 30 pounds gage pressure. In the Public Service Commission tunnels, Levy reports that the carbon dioxid attained a partial pressure of 1 per cent of an atmosphere only upon two or three occasions.

Carbon monoxid is at times liberated in tunnels and caissons through the use of explosives, and has been found to amount to as much as 36 per cent of the gaseous products of combustion. It is an odorless, colorless, highly toxic gas, being dangerous in concentrations as low as 0.15 per cent of an atmosphere. Its affinity for hemoglobin is approximately three hundred times greater than that of oxygen and, in contra-distinction to oxyhemoglobin, carbon monoxid hemoglobin is a relatively stable combination.

Though carbon monoxid has not been regarded as the cause of caisson-disease, it is obvious that the specific action of the gas, with the consequent diminution of the power of blood to take up oxygen, would favor the production of serious cases of caisson-disease.

The removal of carbon monoxid after blasting is a problem of ventilation. The essential treatment of carbon monoxid poisoning is the breathing of oxygen in subtoxic concentrations.

There is some degree of natural ventilation in tunnels and caissons through leakage and the air locks, but it is essential that there be specific reckoning of the quantity of air supplied. The average supply of fresh air per man per hour in the Pennsylvania tunnels was 7,699 cubic feet and in the Public Service Commission tunnels was over 4,000 cubic feet.

Air from a compressor is of such a temperature as to demand some method of cooling before its passage to a working chamber if healthful conditions are to be maintained. Cooling may be effected in reserve tanks or by a system of cold-water pipes about which the air is passed. The air in caissons or tunnels need not rise above 75° F. McWhorter stated that the temperature in the Pennsylvania tunnels was variable, averaging about 75° F. In the Public Service Commission tunnels, although during the periods of construction the external temperature varied from 102° F. to 13° below zero F., the air in the tunnels was practically constant at 77° F.

Humidity in caissons is usually very high, the air being practically saturated. Air movement is limited. Prolonged physical exertion in the warm, moist air of caissons is unquestionably productive of excessive fatigue and its effect upon the heart and the entire vascular system is such as to predispose toward disaster as a result of decompression. Hill maintains that the wet bulb temperature should not rise above 70° F. and that 60° F. is the ideal wet bulb temperature for health and work. He recommends that for use in small caissons the air before compression be dried over calcium chlorid and from the compressors pass for storage to reservoir tanks where under a pressure of approximately 2,000 pounds the water vapor would condense.

In neither the Pennsylvania nor the East River tunnels was there apparent any important positive correlation between temperature or humidity and the incidence of caisson-disease.

Selection of Workers.—Much has been written upon the relation of age to caisson-disease and though, according to Hill, "statistics of caisson and diving works tend to suggest that the percentage number of men affected injuriously by exposure to compressed air increases with age," existing statistical data are not wholly convincing. Hill points out that the variations might depend upon "(1) actual age difference, (2) an increase in mean body weight and fatness with age, (3) on a combination of (1) and (2)."

Pol and Watelle found that men between eighteen and twenty-six years of age best stood caisson work. Keays and Erdman were both of the opinion that young adults were best suited to such work and that men over forty years of age were not desirable. Levy's experience led him to state that advanced age (over fifty years?) may not be of as great importance as it has been considered.

It is a widely accepted belief that fat individuals are ill suited to caisson work. This belief is well supported by experimental evidence regarding the high saturation point of fat for nitrogen and by the relatively slow desaturation of fat by reason of the quantity of gas dissolved and the poor vascular supply of fat tissue.

Boycott and Damant, who conducted extensive studies of the relation of fatness to caisson sickness, conclude that "really fat men should never be allowed to work in compressed air, and plump men should be excluded from high pressure caissons—for example, over 25 pounds—or from diving to more than about ten fathoms, and at this depth the time of their exposure should be curtailed."

In Levy's experience, men with a moderately increased amount of adipose tissue were not more susceptible to compressed air illness than were average individuals.

Organic disease of the heart, lungs, or kidneys is considered adequate cause for rejection of men seeking employment under compressed air.

There is no evidence that immunity to caisson-disease can be established by repeated exposures to compressed air, but there are, however, grounds for belief that certain individuals are for unknown reasons peculiarly unsusceptible to the disease. Hill attaches great importance to the colloid nature of the blood; to the amount of fat in the blood, chyle and the liver, the latter having an enormous capacity for blood; and to fermentation and gas in the intestines. It is his belief that gas in the intestines may serve to furnish "points" for the formation of bubbles of nitrogen. It is upon the basis of such reasoning as this that compressed air workers are urged to avoid fatty foods and to keep the bowels moving actively.

Decompression.—It is excessively rapid decompression from pressure in excess of 15 pounds, gage, which constitutes the greatest hazard of diving or caisson work. With extremely slow decompression it is probable that but very few cases of compressed air illness would result even after long exposure at high pressures. Hill subjected himself to a pressure of 75 pounds, his coworker, Major Greenwood, to a pressure of 92 pounds, both investigators being safely decompressed on many occasions from high pressures. Though it has been demonstrated that exceedingly slow decompression results in no ill effects, it has been necessary to determine rates of decompression from various pressures which will be reasonably safe and at the same time be acceptable to workmen and their employers.

Two methods of decompression are now favored: one known as uniform decompression, during which the air pressure is lowered at a uniform rate; the other, stage decompression, during which the pressure is allowed to fall rapidly until one-half of the absolute pressure is attained, after which decompression is slowly completed at a uniform rate (Haldane).

Under comparable pressure changes desaturation of body tissues will take place in the same period of time as saturation. It has been stated previously that full saturation requires several hours—five hours, in Haldane's opinion. Obviously it is not practicable to subject compressed air workers to a process of decompression so prolonged. In recent years it has been customary to decompress at a uniform rate of 1 to 2 pounds per minute, though stage decompression is becoming more generally used than in the past. The State of New York now requires stage decompression,[3] the total period being determined on a basis of decompression at minimum rates varying from 1 to 3 pounds per minute.

The regulations adopted, respectively, by Massachusetts in 1916, New Jersey in 1914, New York in 1922 and Pennsylvania in 1922 provide for decompression in the following total periods of time:

Decompression Provided by State Regulations

Gage Pressures in Pounds	Total Time of Decompression in Minutes			
	Massachusetts	New Jersey	New York *	Pennsylvania *
0 to 10	1	1	3.3	3.3
11 to 15	2	2	7.5	7.5
16 to 20	5	5	13.3	13.3
21 to 25	12	10	16.6	16.6
26 to 30	15	12	30.	30.
31 to 35	20	15	35.	35.
36 to 40	40	20	40.	40.
41 to 50†	50	25	45.	45.

* Time given relates to maximum pressure indicated in first column.

† The regulations of Massachusetts and New Jersey provide that no person shall be permitted to work under a pressure exceeding 50 pounds except in cases of emergency.

Haldane showed that as much gas would be liberated during decompression from a pressure of four atmospheres to that of two, as from that of two atmospheres to that of one. That is, the quantities of liberated gas in the two instances would be determined by the relative diminutions in pressure. Common experience having demonstrated that no serious damage was wrought by rapid or even instantaneous decompression from a gage pressure of 15 pounds, a reduction of pressure from two atmospheres to one, and no fatal cases after decompression from a pressure of 19 pounds, Haldane argued that "the absolute pressure can always be rapidly reduced with safety to one-half the absolute pressure at which the tissues are saturated," such a partial reduction to be followed by slow decompression, the worker leaving the decompression chamber with a saturation of body tissues equivalent to that of 19 pounds gage pressure.

The table on page 703 presents Haldane's recommendations regarding decompression after exposure to various pressures in caissons or tunnels, the figures in parentheses representing the number of minutes required for total decompression, including the initial rapid stage accomplished in each instance in three minutes.

Japp, of the opinion that the long periods for decompression required under the conditions of the Haldane method were not practicable, devised

---

[3] Providing for a drop of one-half the maximum *gage* pressure at the rate of 5 pounds per minute.

DECOMPRESSION AFTER EXPOSURE TO VARIOUS PRESSURES

Working Pressure in Pounds per Square Inch	Number of Minutes for Each Pound of Decompression after the First Rapid Stage		
	After First 3 Hours' Exposure	After Second or Third 3 Hours' Exposure	After 6 Hours or More of Continuous Exposure
18 to 20	2 (6 to 8)	3 (7.5 to 10.5)	5 (10.5 to 15.5)
21 to 24	3 (12 to 16.5)	5 (18. to 25.5)	7 (24 to 34.5)
25 to 29	5 (28 to 38)	7 (38 to 52)	8 (43 to 59)
30 to 34	6 (48 to 60)	7 (55.5 to 69.5)	9 (70.5 to 88.5)
35 to 39	7 (73 to 87)	8 (83 to 99)	9 (93 to 111)
40 to 45	7 (90.5 to 108)	8 (103 to 123)	9 (115.5 to 138)

a table of decompression rates based upon a maximum saturation of body tissues of 25 pounds upon emergence from the decompression chamber.

RATES OF DECOMPRESSION BASED ON 25 POUNDS MAXIMUM AIR SATURATION OF BODY ON EMERGENCE FROM DECOMPRESSION CHAMBER

Working Pressure in Pounds	Pounds of Pressure to Which Reduced in 3 Minutes	Total Number of Minutes in Air Lock After 8 Hours' Work	Total Number of Minutes in Air Lock After 3 Hours' Work	Total Number of Minutes in Air Lock After 2 Hours' Work	Pounds of Maximum Air Saturation of Body on Emerging
27	6.0	9			25
30	7.5	24			25
32	8.5	33	25		25
35	10.0		35		25
40	12.5		48		25
42	13.5		51	37	25
45	15.0			42	25
50	17.5			48	25

In the East River tunnels, Levy states that decompression was effected by a drop to one-half of the gage pressure, this pressure was maintained "for a certain length of time," then followed by slow decompression to atmospheric pressure.

Hill has recommended that in stage decompression from 30 pounds gage pressure there be a pause of 15 minutes at 10 pounds pressure; in decompression from a working pressure of 45 pounds, a pause of 30 minutes at 15 pounds pressure. He has expressed the belief that stage decompression is preferable to the uniform method for caisson work and that Japp's results (Pennsylvania tunnels) prove that the standards for decompression periods set forth in the Admiralty Committee's report are about twice as long as necessary.

Because of the stimulation of the circulatory mechanism attendant upon exercise, it is of great importance that men undergoing decompression be induced to exercise and that they continue active for some minutes after leaving the air lock. Hill has suggested the inhalation of oxygen during decompression as an additional precautionary measure.

Treatment of Caisson-Disease

The one therapeutic measure of transcendant importance is *recompression* of an affected individual at the earliest moment possible after the onset of symptoms.

Pol and Watelle in 1854 described cures obtained through recompression of workmen suffering with "the bends." Though the value of recompression was experimentally demonstrated by Bert, the first hospital lock was used by Moir at the Hudson tunnels constructed in 1890. Since that time a chamber for the recompression of workmen affected by caisson-disease has become a part of the standard equipment of organizations undertaking engineering operations under compressed air. The state regulations in Massachusetts provide that a medical lock shall be maintained in connection with all work in compressed air; the regulations of New Jersey, New York and Pennsylvania, in connection with all work in compressed air when the maximum pressure exceeds 17 pounds.

The hospital or medical lock is customarily a steel cylinder about 6 feet in diameter and 12 to 18 feet long of solid construction at one end, the entrance at the opposite end being fitted with an in-opening door. A partition erected at the middle of the cylinder provides two compartments, a door in the partition opening toward the inner chamber. The latter is furnished with a cot, an electric heating device, a telephone and necessary medical supplies. Both compartments are connected with a compressed air line and are equipped with proper escape valves, gages, windows and electric lights.

In the series reported by Keays, of 3,692 cases of caisson-disease of varying severity, 3,067 were treated by recompression. The table on page 705 sets forth in some detail the results of Keay's experience.

It should be noted in this connection that only mild cases received medical treatment.

Of 680 cases reported by Levy, all were to some degree relieved by recompression.

Recompression should be instituted promptly after the onset of the disease is evidenced. Keays and his associates were convinced that the benefits resulting from recompression were definitely dependent upon the time elapsing from the onset of symptoms until treatment was received. The table on page 705 is a modification of one published by Keays, an analysis of the results of recompression of 955 cases of pain in one leg.

The state regulations in New York provide that "an identification badge shall be furnished to all employees advising police officials that the employee is a compressed air worker; stating location of medical lock, and stating that in case of emergency an ambulance surgeon shall remove the patient to the medical lock and not to the hospital."

RESULTS OF KEAYS' EXPERIENCE WITH CAISSON-DISEASE

Location of pain	Treated by Recompression				Treated by Medical Means		
	Relieved by one recompression	Relieved by two or more recompressions	Partial relief by recompression	No relief by recompression	Relieved by medical treatment	Partial relief by medical treatment	No relief by medical treatment
One leg	835	238	94	3	88	6	13
One arm	311	104	77	12	43	14	7
Both legs	382	143	55	0	14	1	3
Both arms	54	19	10	0	0	0	1
One leg and one arm	150	53	21	0	7	2	1
Both legs and one arm	64	22	11	0	2	0	2
Both arms and one leg	24	6	5	0	0	0	0
All extremities	73	36	24	0	0	0	0
Abdomen (mild)	124	10	1	0	2	0	0
Scattered and unclassified	80	16	8	2	2	1	2
Total	2,097	647	306	17	158	24	29

Total number of cases treated by recompression, 3,067.
Cases relieved by one recompression, 2,097, 68.3 per cent.
Cases relieved by two or more recompressions, 647, 21 per cent.
Cases relieved by one or more recompressions, 2,744, 89.3 per cent.
Cases partially relieved by recompression, 306, 9.9 per cent.
Cases not relieved by recompression, 17, 0.5 per cent.
Total number of cases treated by medical means, 211.
Cases relieved by medical treatment, 158, 74.8 per cent.
Cases partially relieved by medical treatment, 24, 11.3 per cent.
Cases not relieved by medical treatment, 29, 13.7 per cent.

Despite the existing evidence of the value of early treatment, it may be successfully employed after a lapse of many hours following the development of pain and there are on record cases of recovery attributed to recompression performed several days after the onset of disability.

RESULTS OF RECOMPRESSION OF 955 CASES OF PAIN IN ONE LEG

Time at Which Treatment Was Given after Onset of Symptoms	Number of Cases	Results in Percentage of Cases Treated by Recompression		
		Relieved by One or More Recompressions	Partial Relief by Recompression	No Relief by Recompression
Under 2 hours	639	91.9	8.1	0
2 to 4 hours	89	93.3	6.7	0
4 to 6 hours	99	93	6	0
6 to 12 hours	104	85.5	12.5	1.9
Over 12 hours	24	75.	20.8	4.1

The pressure used in recompression should be the same as that under which the patient has been working and the maximum pressure should be maintained for some minutes before decompression begins. The latter may be effected by either the uniform or the stage method, though the stage method is considered preferable. Keays recommends the use of modified Haldane stage decompression, reducing the pressure quickly to 10 or 15 pounds, then slowly completing decompression, the total period being at the rate of two minutes for every pound of pressure applied. Levy advises using the stage method but doubling the time allowance for each stage of decompression. As in routine decompression, active movement of the subject should be encouraged. If a single recompression does not afford full relief, it should be done repeatedly.

Medical treatment is essentially symptomatic. Massage, hot baths and morphin may be used, as the severity of a case may indicate. Oxygen is probably of value in serious cases.

HEAT, HUMIDITY AND COLD

Heat

Intense heat is not infrequently an industrial health hazard, though modern methods of artificial ventilation and various mechanical developments have materially reduced the exposures in numerous industries.

Smelters, foundrymen, kiln men, roofers, miners, sugar refiners, cooks, bakers and other workers in great variety are at times subjected to high heat. Iron and steel workers may be exposed to temperatures far beyond that of the body, often for fairly long periods. Men who clean the "stoves" attached to blast furnaces work for periods lasting a few moments each in air of temperatures well above that of boiling water, but the heat is dry and the men apparently suffer few ill effects.

Marine firemen until recent years were in many vessels subjected to exceedingly high temperatures. Naval surgeons, whose reports were quoted by Fiske, found temperatures in fire rooms, engine rooms and dynamo rooms, ranging about 150° F. and as high as 204° F. Improvements in marine construction have mitigated the discomforts of those who maintain power in ships, though heat hazards still exist.

Long exposure to intense heat, particularly if it is associated with high humidity and little air movement, is productive of definite disturbances of function. These disturbances are of several types, though individual cases may not be clearly defined. They may be grouped as cases of heat exhaustion, heat stroke, muscle cramps, and those presenting the more superficial effects of radiant heat.

Heat Exhaustion and Heat Stroke.—Fiske, discussing the effects of heat observed by naval surgeons, states:

> "The heat regulation centers do not appear to be so profoundly incapacitated as in sunstroke, there being but slight fever or very commonly the subnormal temperature of shock. . . . There is pallor or lividity, drenching perspiration, weakness of both voluntary and involuntary muscle, the circulatory and respiratory functions being profoundly embarrassed. Sensory motor phenomena are those of exhaustion rather than of the irritable, responsive to the least stimulation, type; there is the tendency to sleep and stupor rather than to convulsions. Now, although paradoxical, it is no inconsistency that the other notable symptoms may become prominent, such as fibrillary twitchings and violent painful cramps of muscles of abdomen, arms and legs."

Sayers and Harrington, in describing the effects of high temperatures and high humidities in metal mines, report:

> "I. In still air . . . with a wet bulb temperature over 90° F. and under 100° F., and with a relative humidity of 89 per cent or higher, the following signs and symptoms were found, even when little or no exercise was taken: (1) Blood-pressure, systolic and diastolic fell rapidly. (2) Body temperature rose, in one case it reached 102° F., and this after less than two hours having been spent in the hot, humid air described. (3) Pulse rate increased and seemed more sensitive to exercise than normally. (4) Perspiration was very profuse. (5) Dizziness was a common symptom, and sometimes was marked. (6) Physical weakness or exhaustion was marked in some cases and present in all. (7) Inability to think quickly or accurately was a very common symptom. (8) Nausea was occasionally found. (9) Headache was also occasionally found. (10) Loss of weight was especially marked in men who had been employed under the above conditions over a period of years, but occurred even after exposure of only a few days.
>
> "II. In still air, with wet bulb temperatures of from 85° F. to 86° F. and a relative humidity of 96 per cent, there were no marked changes in the blood-pressure or body temperature, nor were the symptoms dizziness, physical weakness, and inability to think or act quickly, mentioned in I, found as long as the subjects remained at rest or took only light exercise. When moderate exercise was taken—climbing up and down an eight-foot ladder fifteen times in five minutes—the blood-pressure and body temperature rose somewhat.
>
> "III. Blood-pressure readings taken after the subject had reached the cool air of the surface were found to vary considerably with men unaccustomed to high temperatures. Under conditions which resulted in a rise of body temperature to 100° F., or more, the systolic pressure fell,

but where the conditions were such as not to cause the body temperature to rise above 100° F., there was a rise in the systolic pressure when the subjects reached the surface. In one man, long accustomed to hot, humid air, a fall of systolic pressure was also found. In three others, not accustomed to the conditions mentioned, there was a rise of systolic pressure.

"IV. It was found that the body temperatures reached normal in from one to two hours after the subjects had reached the cool air of the surface after having been subjected to conditions that caused a rise above 100° F."

Heat exhaustion as above described presents a clinical picture quite different from that of heat stroke or sunstroke. In the latter conditions the heat-regulating mechanism of the affected individual seemingly is unable to respond to the burden thrust upon it and the body temperature rises rapidly. Headache, dizziness, nausea, vomiting and diarrhea may be premonitory symptoms. Transitory loss of consciousness or coma may ensue. In certain cases death is abrupt. The temperature may be very high, from 103° to 109° F. or even higher in cases usually fatal, the face flushed, the pulse at first rapid and bounding, later irregular and of little force. Breathing is at first deep, possibly stertorous. In severe cases the respiration becomes shallow and may be of the Cheyne-Stokes type. The pupils may be dilated early, later becoming contracted.

On postmortem, the left heart is usually found well emptied, but the right ventricle filled with blood; the lungs are congested, and degenerative changes are observed in the kidneys.

The prognosis in heat stroke is fair for non-alcoholic individuals of previous good health, especially if treatment is instituted promptly after the onset of symptoms, though a mortality rate of about 25 per cent is reported for groups of hospital cases. Following heat stroke, impaired memory and mental function may persist for months. Victims of heat stroke or of heat exhaustion in many instances are for years or permanently particularly susceptible to the influences of even moderately high temperatures.

Treatment.—The treatment of heat exhaustion is usually simple. If the temperature is elevated above 102° F., cooling sponges may be given until the body temperature is reduced to 102° F. If the temperature is subnormal, warm baths may be given and mild stimulation offered, such as hot drinks or aromatic spirits of ammonia. If depression is marked camphor, caffein, ammonia or nitroglycerin may be used. Bromids are of value in cases showing nervous irritability and hot baths or morphin may be demanded for the control of convulsions. Antipyretics should be used with caution because of their depressing effects. As a rule, rest in a moderately cool room suffices, and recovery is complete within a day or two.

Heat stroke calls for prompt and active treatment. The patient should be stripped, placed in a cold tub and vigorously rubbed with ice. Cold water played upon the body and the head from a height of several feet offers additional stimulation. Ice packs and ice water enemata are also recommended. These measures should be abandoned when the temperature reaches about 102° F. The condition of the heart and pulse should be observed and cardiac stimulation given if needed. Bleeding may be of great value in cases threatened with asphyxia from pulmonary congestion and should be done promptly if edema of the lungs is evident.

Heat Cramps.—Edsall in 1904 described in detail cases of transitory myokymia and myotonia induced by exposure to heat. The condition somewhat resembles tetany, but it cannot be identical with that disease. It is clearly distinct from typical heat stroke and may be recognized without association with the characteristic symptoms of heat exhaustion, though, in certain cases, it is likely associated with some degree of exhaustion. Marine surgeons have for many years observed cases of this nature developed among stokers, and cases have also been reported in iron and steel workers and other persons exposed to high heat.

According to Edsall, the patient is quite suddenly seized with spasms of various groups of muscles. The fingers may be first involved, then the calves, the thighs, the forearms and the muscles of the abdominal wall. At the onset the patient is flushed and may have a slight fever. The muscle movements are described as twitching or writhing, the spasms as of short duration but recurrent. In the intervals between severe attacks the muscles are in tonic spasm and may show fine fibrillary movement. At such times spasms may be induced by even slight stimulation, such as palpation of the muscles or gentle brushing of the skin. There are no sensory changes and the superficial reflexes are not exaggerated.

Spasms do not usually occur spontaneously after about twenty-four hours, though they may be induced by stimulation for one or two days longer. The cases may be severe and even result fatally, due possibly to cardiac spasm. The severity of a case seems to be dependent upon the temperature to which the individual is exposed, the duration of the exposure, and the degree of muscular activity.

It is Edsall's opinion that the condition is not due to dehydration of the tissues but is, rather, of toxic origin, associated with an acute degenerative process in the muscles. The pain of spasm of abdominal muscles should be differentiated from intestinal cramps induced by the drinking of cold water.

Treatment.—Treatment of heat cramps is essentially symptomatic. Rest, tepid or warm baths, and mild sedatives may be used. Many naval surgeons, believing that the muscle spasms are in some degree due to dehydration, exert every effort to force fluids upon affected individuals.

Miscellaneous Effects of Heat.—Radiant heat to which such workers as bakers, furnace men, glass blowers, molders and stokers are exposed may produce a variety of skin lesions. Prosser White describes "marblings" of the legs of such workers a congestive reaction to heat which may be followed by vesiculation and pigmentation similar to that produced by sunburn. Burns of the second degree, involving extensive inflammation but not ulceration, may be produced by radiant heat. They should be treated as sunburn, "whitewash" being a very effective agent.

Vision may be severely affected. Watkins states that conjunctivitis and blindness are common among men exposed to intense radiant heat. The cataracts produced in the eyes of glass blowers, puddlers and tin-plate workers are considered to be due to the action of infra-red or heat rays. This subject is discussed elsewhere in this chapter.

In addition to the definite and specific effects of heat, there are various chronic conditions ascribed to long-sustained exposure to high temperatures. Swollen and tender feet may result from continued standing on hot surfaces, such as the metal plates about many types of furnaces. Arthritis, arteriosclerosis and kidney disease are also associated with exposure to heat, though in many cases it is impossible to establish beyond doubt the causal relation of the exposure to the clinical pictures presented.

Prophylaxis.—Much may be accomplished, in the direction of eliminating or reducing industrial heat hazards, through proper construction and arrangement of furnaces, boilers and other sources of heat; through the use of protective clothing and goggles and screens of metal, asbestos or water; and through proper ventilation secured as natural draft, by exhausts or by air blasts directed from the exposed worker toward the sources of heat.

It is highly desirable that an adequate supply of drinking water be readily accessible. The temperature of the water should be about 55° F. and never cold. Workers should be encouraged to drink small quantities at frequent intervals. There can be little doubt of the fact that dehydration is an important factor in many cases of heat exhaustion.

Men employed at work involving exposure to intense heat should, in the selection of food, limit the amounts of fat and protein eaten.

It is, above all, important that the duration of active work under high temperatures should be determined in relation to the severity of exposure. Work which could not be safely undertaken over a long spell may be performed without ill effects if done in short spells alternating with periods of rest.

HEAT AND HUMIDITY

The effects upon the organism of high temperatures should not be considered apart from the question of humidity. Temperatures well above 100° F. can be safely borne if the air is dry and in motion, but a wet bulb temperature of about 78° F. or above represents unsafe working conditions. The body not only gives off heat by conduction, radiation and convection, through expired air and with the excreta, but also through evaporation of perspiration.

Heat loss is controlled by the regulating mechanism of the body, centered in the medulla, the peripheral circulation being altered in accordance with variations in the external temperature. Although this response to varied atmospheric conditions is maintained within the limits usually encountered, above a wet bulb temperature of 78° F., heat loss is not sufficiently rapid to enable an individual to engage in muscular activity without a rise in body temperature, particularly if air movement is slight.

High wet bulb temperatures are encountered in certain industrial establishments such as textile mills, laundries, tanneries and in many mines. Work places such as laundries and some mines are of necessity or naturally damp, but the humidity in textile mills is artificially maintained.

But few efforts have been made by governmental agencies to control the problem of humidity in industry. The labor laws of Massachusetts prescribe permissible limits of wet and dry bulb temperatures, and in Great Britain it is required that the difference between wet and dry bulb temperatures shall never be less than 2° F., with a few exceptions toward the upper limits of permissible temperatures. Artificial humidity shall not be used when the wet bulb temperature exceeds 75° F.

Leonard Hill, recognizing that the dry bulb thermometer was a "static instrument" not showing the evaporating power of air, that "the body is dynamic, producing heat which must be lost at an equal rate," devised the kata-thermometer, "a large-bulbed spirit thermometer of standard size and shape, graduated between 100° and 95° F. The bulb is heated in hot water in a Thermos flask until the meniscus rises into the small top of the bulb. It is then dried, suspended, and the time of cooling from 100° to 95° F. taken with a stop watch in seconds. The number of seconds divided into a factor number (approximate to 500 and determined for each instrument) gives the cooling power by convection and radiation exerted on the surface of the 'kata' at approximately skin temperature in millicalories per square centimeter per second. The operation is repeated with a moist cotton muslin finger stall on the bulb and the wet 'kata' cooling power obtained, the cooling power due to evaporation, radiation and convection. The difference between the two gives the evaporative cooling power." The instrument is very sensitive to even slight air movement and offers a more trustworthy index of conditions favoring or not favoring comfort than the wet bulb thermometer. As pointed out by Harrington and McElroy, even "the physical condition of an individual, his natural tendency to perspire, his clothing and his degree of muscular activity may be determining factors in the estimation of values of surrounding air, as they concern comfort."

Orenstein and Ireland present the following table, based on observations upon natives employed in the Rand mines:

PHYSIOLOGICAL EFFECTS OF WET AND DRY KATA ON MAN STRIPPED TO THE WAIST

Cooling Power		Effects
Wet Kata	Dry Kata	
5	1.5	Extremely oppressive, profuse perspiration, rise of body temperature and pulse rate, especially when work is performed. Very small evaporation from the respiratory tract.
10	3.5	Distinctly oppressive, body temperature can be kept nearly normal only by profuse perspiration. Skin flushed and wet. Pulse rate high.
15	5.5	Lower limit for comfort.
20	8.0	Quite comfortable for work.
25	10.0	Cool and refreshing for work, too cold for resting, especially after being heated.

They further estimated the relative efficiency of workers under varying conditions of dry cooling powers.

Dry cooling power..................	1	2	3	4	5	6
Working efficiency (per cent).......	50	60	70	80	90	100

Representative findings of the U. S. Bureau of Mines and the U. S. Public Health Service in the course of investigations in metal mines in this country are presented by Harrington and McElroy in the table on page 713.

Estimation of degrees of comfort have been correlated by the same authors with dry and wet kata cooling powers. They apply to persons wearing moderately heavy clothing, practically at rest.

Generally speaking, comfort demands that if workers are but slightly active the dry kata cooling power should not fall below 6, or the wet kata cooling power below 18; if they are moderately exerting themselves, the dry kata power should not fall below 7, the wet kata power below 20.

In a report of the British Health of Munition Workers Committee are found the following illustrations of favorable and unfavorable working conditions, certain of which could not be determined from dry bulb or wet bulb thermometer readings alone, but which are clearly indicated by the kata cooling process.

Representative Findings in U. S. Metal Mines

Mine	Place	Air Motion (Feet per Minute)	Temperature			Cooling	
			Wet Bulb Degrees F.	Dry Bulb Degrees F	Per Cent Relative Humidity	Dry Kata	Wet Kata
A	Underground	None to 1270	67.0 to 73.5	69.5 to 81.5	84 to 98	3.10 to 24.4	6.92 to 29.6
B	Underground	None to 825	72.0 to 82.0	76.0 to 86.0	74 to 96	3.11 to 11.0	7.62 to 19.0
C	Underground	None to 100	68.0 to 84.0	73.0 to 85.0	61 to 98	2.34 to 6.52	5.80 to 15.7
D	Underground	None to 185	67.5 to 91.5	77.0 to 91.5	62 to 100	0.61 to 7.58	2.42 to 18.4
E	Underground	None to 810	37.0 to 59.0	39.0 to 62.0	77 to 100	6.44 to 26.8	None taken
F	In change-house	None	57.0 to 69.0	72.0 to 82.0	40 to 70	2.9 to 9.2	10.8 to 17.1
	Surface air	None to slight	33.0 to 64.0	33.0 to 68.0	52 to 100	6.2 to 28.3	15.4 to 42.5
	In shaft	None to slight	52.0 to 57.0	52.0 to 57.0	96 to 100	8.8 to 11.1	16.4 to 19.6
	Underground	None appreciable	52.0 to 67.0	52.0 to 67.0	90 to 100	5.9 to 7.5	11.8 to 18.9

Conditions with Dry and Wet Kata Cooling Power

Condition	Dry Kata	Wet Kata
Very uncomfortable	Below 2.5	Below 7
Slightly uncomfortable	2.5 to 3.5	7 to 9
Barely uncomfortable	3.5 to 4.0	9 to 10
Just comfortable	4.0 to 5.0	10 to 12
Quite comfortable	5.0 to 7.0	12 to 15
Very comfortable	7.0 to 10.0	15 to 20

WORKING CONDITIONS INDICATED BY THE KATA COOLING PROCESS

Conditions	Wet Bulb	Dry Bulb	Wet Kata	Dry Kata	Evaporative Power
Bright, pleasant day in May, out of doors	60	68	27.2	7.5	19.7
Brass foundry (good)	60	72	24	7.3	16.7
Machine shop (bad)	61	72	15	4.6	10.4
Cartridge annealing and cleaning (bad)	64.5	80.5	17.5	3.0	14.5
Cartridge annealing and cleaning (good)	54.5	60	24.	9.0	15.0

A desirable atmosphere should be: "(a) cool rather than hot; (b) dry rather than damp; (c) diverse in temperature in different parts, and at different times, rather than uniform and monotonous; and, which is intimately connected with this diversity; (d) moving rather than still."

COLD

A relatively small number of industrial workers are, by the nature of their occupations, exposed to very low temperatures. Outdoor workers, such as chauffeurs, sailors and street cleaners, may in northern regions be obliged to spend many hours daily in cold air. In addition to these, employees in cold-storage plants, fish wharves, slaughterhouses, ice plants or storehouses, ice-cream factories and a variety of other establishments are subjected over long periods to cold and usually, also, to dampness.

The effects of cold are varied. Chilling may result in the lowering of body resistance and thus in increased susceptibility to respiratory infections. The aggravating influence of cold damp air upon arthritis is productive of joint pain in many members of this group. There are, however, beside these contributory effects, a number of specific clinical conditions which are in many cases directly attributable to cold. It has long been thought that nephritis could be induced by exposure to cold, and chronic nephritis is said to be common among workers in cold places. Gaisbock has produced glomerulotubular nephritis in rabbits by direct chilling of the kidneys. Cicconardi, using rabbits, found that exposure to cold, applied locally to the kidneys or by immersion, produced a transient nephritis, with degenerative changes in the tubules, albuminuria and casts. Widal and his associates were of the opinion that the condition produced in dogs by immersion in cold water was identical with that produced by the intravenous injection of foreign protein and with anaphylactic shock.

Paroxysmal hemoglobinuria in clinical experience is frequently related definitely to exposure to cold, and may be produced experimentally. Little is known, however, of the nature of the processes which are involved in this disease. The treatment of hemoglobinuria and of nephritis is considered elsewhere in this work.

Localized neuritis not infrequently results as an effect of cold. A common example is found in the neuritis involving the seventh cranial nerve (Bell's palsy).

A study of the "white fingers" of stonecutters using pneumatic tools, made in 1917 and 1918 by Alice Hamilton, Edsall and others, led to the conclusion that the spastic anemia associated with loss of sensation in the hands, while due to the use of the rapidly vibrating tools, was elicited by exposure to cold.

Many of the effects of cold are especially manifested when there is alternation of exposure to cold and heat, a circumstance often encountered in such establishments as storage warehouses where workers go from the cold interior to heated outdoor air, passing out and in repeatedly.

Vasoconstriction is produced in exposed parts of the body by chilling, and, when cold is succeeded by warmth, dilation results with possibly congestion, edema, redness, sensory changes and some degree of vasomotor paralysis. Severe chilling may lead to occlusion of small blood-vessels, with ensuing tissue destruction.

Chilblains are common effects of cold, characterized by vasoconstriction, paresthesia and usually persistent sensitiveness to cold. With frostbite there is edema, vesiculation, desquamation and possibly deep ulceration.

"Trench foot" is a disease essentially of the same origin, resulting from severe vasomotor disturbance. It is fostered by constriction of the muscles of the leg and foot and by perspiration.

The scleroderma of stonemasons, described by Bramwell, is apparently somewhat analogous in its origin to the spastic anemia of stonecutters hitherto described.

Prophylaxis and Treatment.—The use of adequate clothing is of first importance in the prevention of the effects of exposure to cold. When possible, moisture in cold air should be eliminated or reduced, and strong air currents avoided.

The essential principle in the treatment of superficial manifestations of the effects of cold is the avoidance of the use of any measure which induces an abrupt reaction in affected parts. Frost-bitten members should be rubbed with ice or snow or in cold water, not only as a first treatment but on succeeding days. Surgery is indicated when gangrene develops, but only after a line of demarcation is established.

LIGHT

Light is believed to be a wave form of energy related to Roentgen rays and to the hertzian waves utilized in wireless telephony and telegraphy. These waves are supposed to consist of "vibrations of the ether" propagated through space at a rate of 186,000 miles a second (3×10^{10} cm. a second). Some writers assume the vibrations to be of the nature of rhythmical alternations in electromagnetic intensity. For the present purpose the wave hypothesis is adequate, as it is the relation of the waves that is to be discussed. Wireless waves are of great size and vary from many hundred meters to fractions of a meter in length; in the case of shorter waves, the frequency of vibration is increased in order to maintain the speed of travel. At a wave length of 0.00076 mm. expressed as 760 μμ or 0.76 μ,[4] where the rate of vibration is about 400,000,000,000 times a second, this energy is perceived by the eye as light of a dark red color. With increasing rapidity of oscillation and a shorter distance between the crests of the waves, the eye experiences successively the sensation of the various colors of the spectrum; through red, yellow, green, blue, and violet until, with a vibration rate of 800,000,000,000 times a second and a wave length of about 300 μμ, the end of the visible spectrum is reached.

It is possible to study and make use of waves of length other than those ordinarily visible. Those greater than 760 μμ are known as infra-red or heat rays; they are given off by objects heated to a dull redness or even below the point of glowing, and are felt as heat. On the other hand, waves, which lie in the spectrum beyond the violet and are shorter than the visible rays, are called ultraviolet. Sometimes they are called chemical rays; their action is bactericidal and produces changes in the silver salts used in photography. For this reason they are known as actinic rays. The concept light covers a great range of values, which are roughly divisible into three main groups, each quite specific in properties and effects. The following table illustrates the approximate relationship:

100 μμ		400 μμ					700 μμ		2300 μμ
X-rays	Ultraviolet Actinic	Violet	Blue	Green	Yellow	Orange	Red	Infra-red Heat	Hertzian Waves
	Invisible			Visible				Invisible	

---

[4] For very small measurements the micron (μ = 0.001 mm.) and the millimicron (μμ = 0.001 μ = 0.000001 mm.) are used. Light is technically measured in Angström units. One Angström = 1×10^{-8} cm.

There is no known pure light of a single wave-length. Light from any source when passed through a prism, which refracts differently the waves of different lengths, can be broken down into its spectrum and analyzed. The effect on the eye is composite. Visible light is called yellow, red, or blue, according to the predominating intensity of the various parts of the spectrum. Similarly pathological effects depend upon the intensities of the various parts of the spectrum, visible, infra-red, or ultraviolet. Much work in analyzing different sources of light has been done especially in the field of illumination. The analyses of light from some ordinary sources follows:

Sunlight.—Continuous spectrum except for Fraunhofer lines to 291 μμ. This lower limit is determined by atmospheric absorption and is identical in all parts of the world.
Quartz; Mercury Lamp.—Discontinuous spectrum with a number of bright lines from 230 μμ to 579 μμ. There are some weak lines between 185 and 230 μμ. Lines below 185 μμ are absorbed by Quartz.
Electric Sparks.—Discontinuous, covers the general field of mercury lamp.
Carbon Arc.—Band spectrum rich in near ultraviolet, weak below 300. No waves below 240 μμ.
Iron Arc.—Line spectrum almost continuous as far as 230 μμ.
Electric Light (Osmium Tantalum).—Spectrum from 300 μμ to 630 μμ.
Incandescent Gas.—320 μμ to 600 μμ.
Petroleum, Kerosene.—350 μμ to 600 μμ.

The most common exposure to light is the universal exposure to sunlight, of which more will be said later. Another almost universal exposure is to light from artificial illuminating sources; but this light—with the exception of the arc light, or the quartz lamp in its scientific and therapeutic uses—in so far as it properly illuminates can hardly be considered pathogenetic. All such sources are of relatively low brilliance or are rendered innocuous through distance, glass, or some other factor. There are, however, a number of occupational exposures to light of great intensity, such as the exposures of iron workers, furnacemen and foundrymen, and glass workers to the light of molten masses; exposure to acetylene and oxyhydrogen welding; and finally exposure to the electric arc, either in the process of welding or in the form of sudden flashes caused by short circuits or blown fuses. The temperatures attained in many industrial processes have been studied, and in some, for example, glass furnaces, the light has been analyzed. On the other hand, where the exact spectra are not known, assumption is made according to a general law that the higher the temperature and the more nearly white-hot the process, the greater the prevalence of short ultraviolet or chemical rays.[5]

---

[5] Glass furnace; T, 2300° F.; spectrum, 340 μμ to 780 μμ infra-red strong. Electric arc; T, 6000° F.; 230 μμ to 600 μμ ultraviolet strong.

The rays of wave-length longer than 760 $\mu\mu$ have a distinct pathological effect as given off by red-hot metals in great abundance. They penetrate the body and act upon the skin as heat, producing a redness and a sensation of warmth which yield quickly to cooling by air. Local metabolic processes are increased according to the thermal coefficient of chemical reactions; but, unless the skin is cooked or blistered by excessive heat, the changes are transitory and no pigmentation results. On the eyes the effect is not noticed, unless the heat is so intense that it is not relieved by lacrimation or winking, in which event burns of the conjunctiva and cornea will result. An after effect of a different sort is the formation of cataracts after long exposure.

GLASS WORKERS' CATARACT

Cataract as a disease of glass workers seems to have been mentioned first by Beer in 1817. Statistical studies were made in Germany by Mayrhoefer in 1886 and Roehlinger in 1888, and in England by Robinson in 1903. All of these reported an incidence of cataract among the bottle finishers of certain districts, far beyond that of other industrial groups. This was disputed and led to an extensive study by Legge for the British Home Office which confirmed the reports of Robinson and others. The cataract affects the finishers especially, but also older men and those who have worked many years.

The cataract has certain characteristics in structure and development. It begins at the posterior cortical layer of the lens, usually at the pole, and in its growth follows the capsule. It is often disc or saucer-shaped, although sometimes it spreads irregularly. The other media of the eye and the retina are not affected, and after extraction of the cataract many men return to work—some of them even as finishers. It is generally agreed that the lesion is caused by looking into the glare of the furnace from which the molten glass is drawn. Robinson suggested that the eyes could be protected by glasses or by screening the furnace. He considered that the central character of the lesion was due to contraction of the pupil and concentration of light and heat rays on the posterior surface of the lens.

Experiments were made to determine the cause of the disease. The eyes of animals were exposed to long-continued action of light, especially light rich in ultraviolet rays, but no definite clouding of the lens could be obtained. A full report of these experiments and a discussion of the literature were made in 1913 by Hess and by Parsons at the Seventeenth International Congress of Medicine (Ophthalmological Section) in London. The most recent summary of this work and the most authoritative present-day opinion is probably embodied in the Report of the British Cataract Committee. They believe that, although some changes can be produced in the lens by ultraviolet rays (chiefly in the region between 320 and 400 $\mu\mu$, the shorter waves being absorbed by the conjunctiva and cornea), nevertheless the causative agent is heat energy; and, as protection against this, they recommend the use of Crookes' glass. More recently a similar condition has been shown to exist in chain makers, puddlers, and tin-plate millmen in Great Britain.

Prophylaxis and Treatment.—*Treatment* of the condition once developed is only through extraction of the opaque lens, after which the eye fitted with spectacles functions well. Yet the disease is quite preventable by goggles of proper glass which permit the passage of light adequate to detailed vision but exclude the heat-producing infra-red rays. It should be added that, on account of the fogging of the goggles from condensation, it is also necessary to furnish workmen with one of the various "antifog" compounds or soaps.

The preparation of protective glasses was undertaken by Sir William Crookes, who determined the temperature, that character and intensity of the light given off from the furnaces. The temperatures ranged from 2,000° to 2,300° F. and the spectra varied with the minerals used. The general quality of the spectra he found to be as follows:

Exposure of Photograph Plate	*Character of Spectrum*
20 minutes	542 to 690 $\mu\mu$
60 minutes	364 to 760 $\mu\mu$
180 minutes	334 to 780 $\mu\mu$

This showed that the ultraviolet rays were of feeble intensity and required long exposure while the heat rays were very powerful. By addition of various metallic oxids to glass and study of the transmitted light, he was able to make various kinds of glass which he considered satisfactory. An example is the Crookes' glass with the following formula:

Fused soda flux	96.8 per cent
Ferrosoferric oxid	2.85 per cent
Carbon	0.35 per cent

This is a pale blue glass which cuts off 96 per cent of heat waves while transmitting 40 per cent of the light. There are numerous others, Crookes' sage-green, Ferrous No. 30, besides many proprietary kinds of glass made in Germany and this country. The U. S. Bureau of Standards has recently tested a number of them for permeability to visible rays and to various parts of the invisible spectrum, and tabulated their findings in a report from which the following diagram is taken:

In addition to these glasses, screening and modern methods of machine glass blowing will contribute to the eradication of the disease by increasing the distance between the worker and the molten mass.

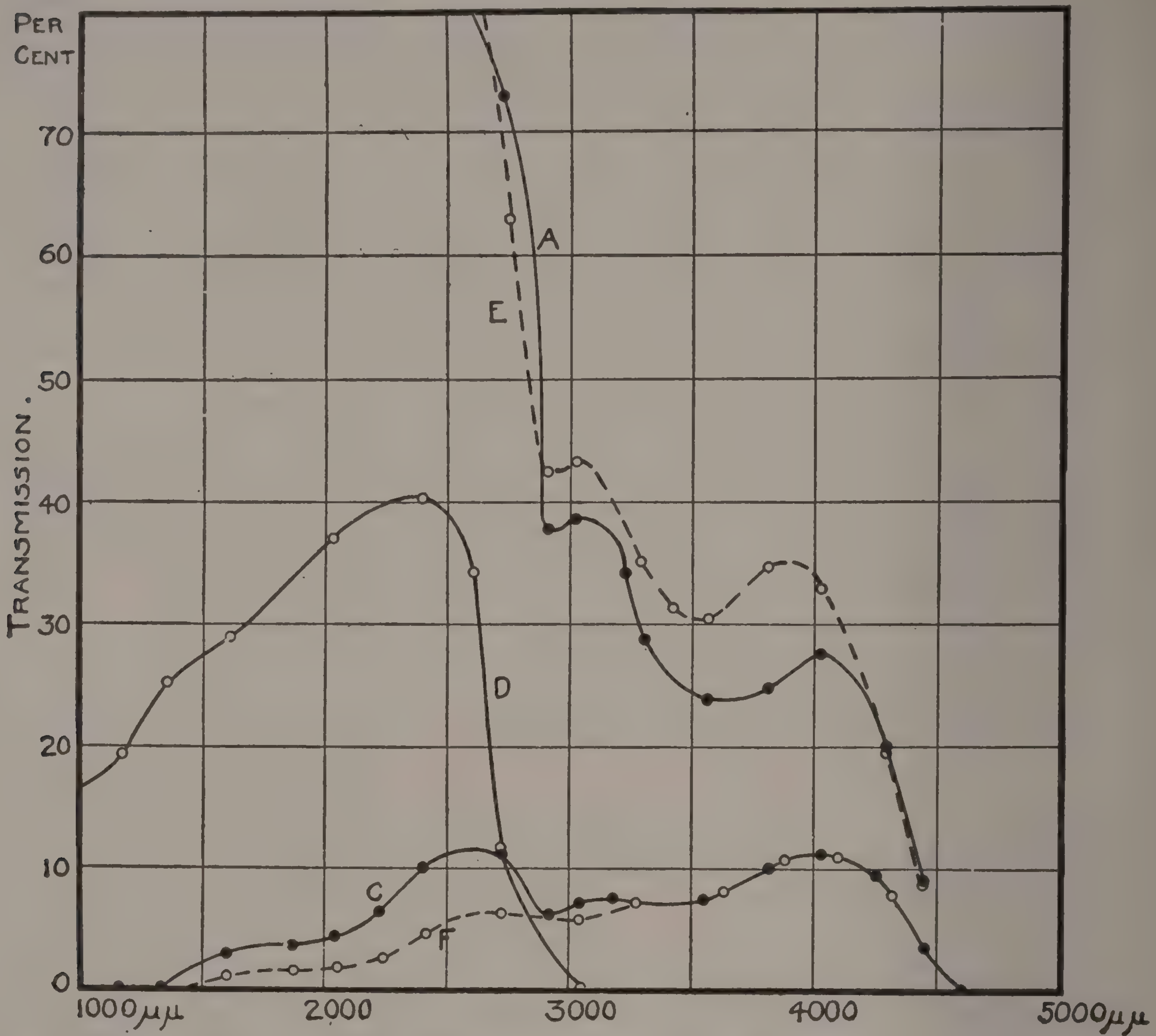


Fig. 1.—Percentage Transmission of Various Glasses for Infra-red Radiation. (After Coblentz and Emerson.) A. Crookes' light neutral (t = 1.96 mm). C. Crookes' ferrous No. 30 sage green (t = 1.98 mm). D. Schlotts' black glass (t = 3.6 mm). E. White crown glass (t = 2.18 mm). F. American Optical Co. Lab. No. 59 (t = 1.93 mm).

Electric Ophthalmia

The acetylene and oxyhydrogen blowpipe are also sources of heat and light energy, but these have so far not been incriminated in causing pathological conditions other than mild conjunctivitis. This is possibly because the heat exposure is relatively short, or on account of the slight intensity of the pathogenetic parts of the spectrum—although there are some feeble ultraviolet rays given off. But in electric welding, in addition to an intense glare, there is strong exposure to the invisible rays at the violet end of the scale. The temperature of the electric arc used in welding is over 6,000° F. and at this temperature the metal gives off a great number of short-length radiations.

Even a brief exposure to this light produces in the eyes a condition known as electric ophthalmia. This was known as early as the middle of the nineteenth century to persons working with the ordinary electric arc at close range. At present it is not uncommon in those exposed to short-circuit flashes, such as motormen, electric power-house hands and linemen. It is in all probability the same condition that produces the ophthalmia of moving-picture actors who work in the glare of a strong arc light, and who, in order to "register" their ocular expression, must expose their eyes to the full effect of the ultraviolet rays.

The symptoms of electric ophthalmia are delayed. A half hour or more following exposure there develops a scratchy feeling of the conjunctivæ; later redness, swelling, and, in severe cases, intense pain. Photophobia is constantly present and persists; contraction of the color field and temporary blindness have been reported.

Treatment.—In general under appropriate treatment the inflammation subsides, although ulceration and sloughing of conjunctiva and cornea may occur. The treatment is that of other forms of severe conjunctivitis.

Ultraviolet Rays

The effect of the ultraviolet rays on the eye has been studied on animals and by self-experimentation. It has been found that the media of the eye do not readily permit of penetration by these rays. In general the energy of the short wave-lengths is transformed in the cornea and conjunctiva and sets up a local irritation; although wave-lengths in the zone of 320 to 400 $\mu\mu$ can reach the lens and in animals cause fluorescence. No retinal changes have been produced. Erythropia (red vision), color changes in the central field of vision, and temporary blind spots have been described by human subjects.

The action of the electric arc on the skin is similarly definite. Although there is no sensation of heat at time of exposure, in from one-half to two hours afterwards erythema develops. No protection is afforded by ordinary or even heavy clothing. The resultant erythema may be severe and lead to vesiculation and ulceration; usually it fades slowly and leaves the skin tanned as after sunburn.

The analogy to sunburn is readily understood, since only the ultraviolet rays in sunlight are effective in producing sunburn. Sunlight analyzed in various parts of the world presents a constant spectrum and has no rays below a wave-length of 291 $\mu\mu$. However, at different seasons of the year the intensity of the several parts of the spectrum (and especially the ultraviolet) varies greatly. This variation has been studied by Huldschinsky, Hess, Howland and others in their investigations of the curative value of sunlight in rickets. More interesting is the work of Dorno who has recorded his own observations and measurements at Davos, Switzerland.

The following charts modeled on his work show the seasonal variation in intensity of ultraviolet rays as compared with the total intensity of the sun's light and heat throughout the year.

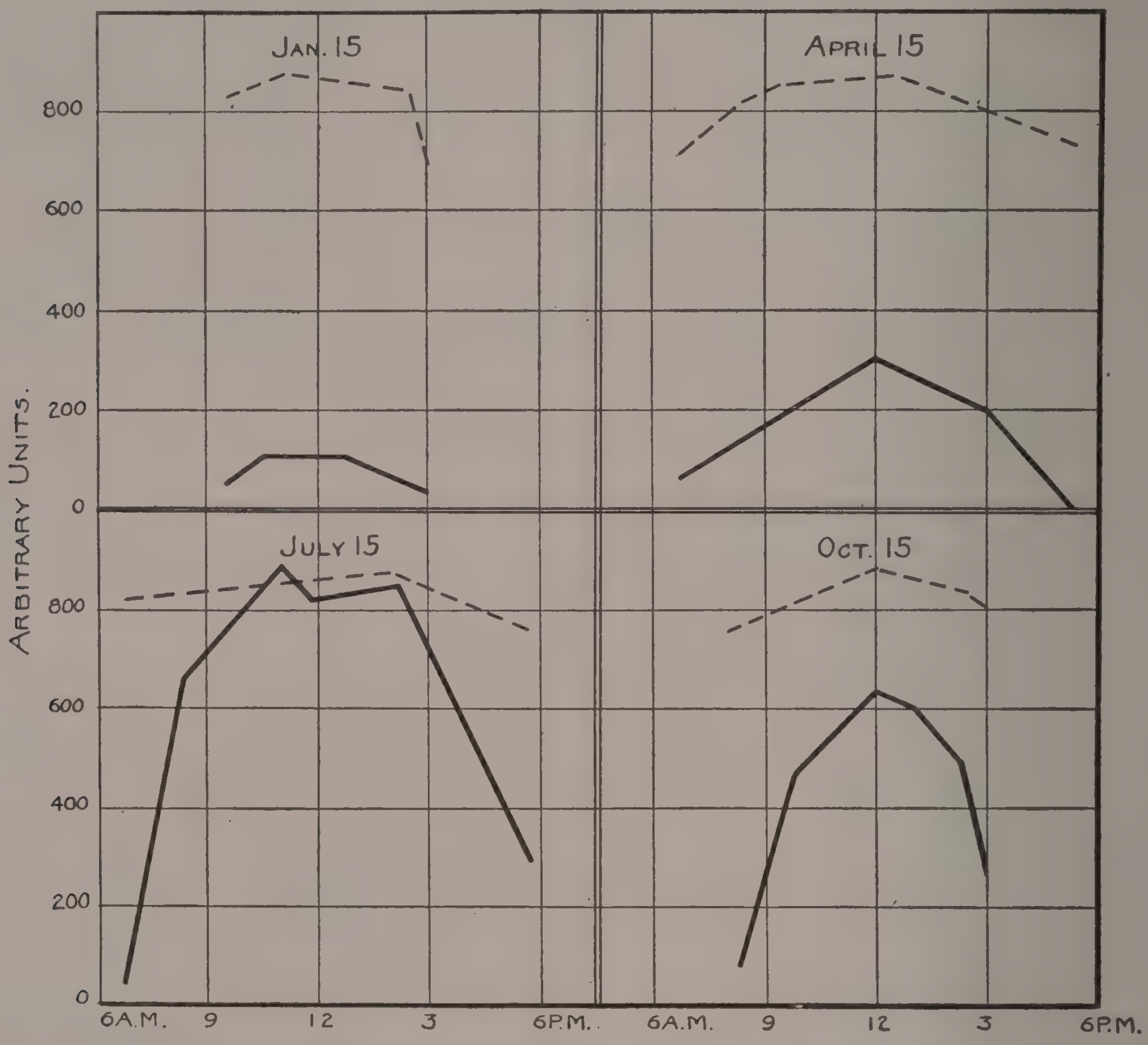


Fig. 2.—Variation in Intensity of Solar Heat (Dotted Line) and Intensity of Ultraviolet Radiation (Solid Line) Throughout the Day in Different Months —in Terms of Arbitrary Units. (After Clark.)

In addition to erythema, sunlight, like the electric arc, produces conjunctivitis and photophobia. Direct sunlight, or glare reflected from brilliant surfaces of water or snow, causes the "snow blindness" from which arctic explorers and mountain climbers suffer. Ordinarily the strong light from the illuminated surface dazzles and prevents long exposure of the eyes; but when the light is not brilliant, as in mist or fog, and the traveler feels the protection of his tinted glasses is no longer needed—under these circumstances snow blindness is apt to occur. The Eskimos protect themselves by a kind of bone goggle with transverse or cruciform slits. The opaque material cuts down the glare; but although diminishing the intensity of the harmful rays it does not really exclude them. The only real protection is by glass suitable to the conditions of exposure.

The ultraviolet effects on eyes and skin are produced by short wave rays from all sources, among which are the various arcs such as iron and carbon, and the quartz mercury vapor lamp. In this latter the quartz permits the passage of waves as short as 185 μμ.[6]

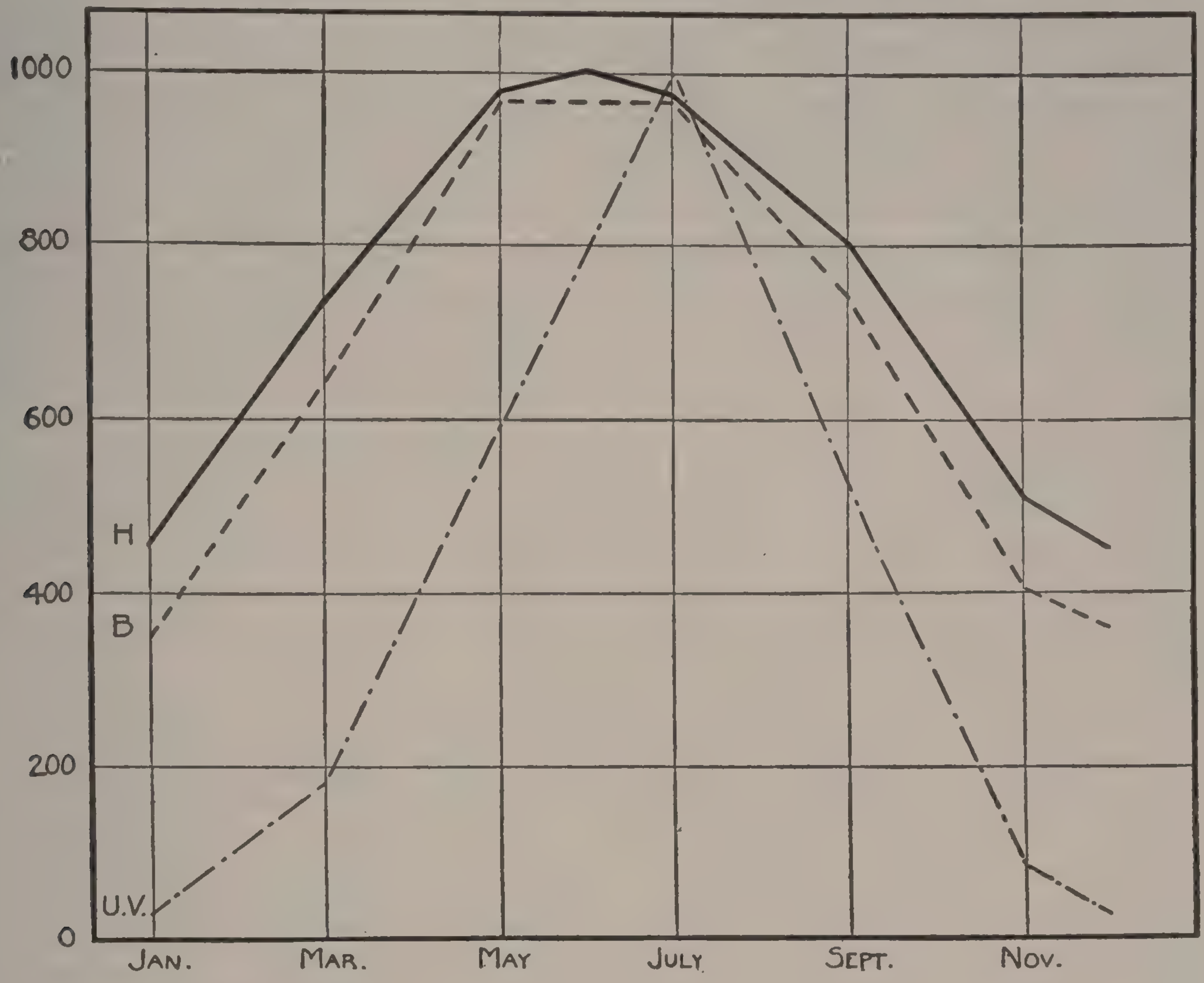


FIG. 3.—HEAT (H), BRIGHTNESS (B), AND ULTRAVIOLET (U. V.) INTENSITIES AT NOON THROUGHOUT THE YEAR AT DAVOS, SWITZERLAND. (After Clark.)

Another difference between the arc and the quartz lamp utilized in light treatments is that the amount of heat given off by the latter is slight. The temperature of the surrounding air rises only a few degrees. In addition there is a strong smell of ozone. The ultraviolet rays

---

[6] The ordinary mercury vapor lamp, as used in illuminating and photography, is enclosed in glass which effectively retains most of the rays of short wave-length. Below is given a table of permeability of various translucent media to ultraviolet rays.

Medium	*Shortest wave-length not absorbed*
Fluorite (2 mm.)	125 μμ.
Crystalline quartz	150 "
Rock salt	177 "
Fused silica	180 "
Uviol glass	280 "
Soda glass (2 mm.)	330 "
Water	190 "
Air	280 "

do not penetrate the skin deeply, though they may do so if the tissues are made bloodless by pressure. Usually their energy is expended on changes in the surface cells and on the blood. The blood becomes photo-active and remains so for some time. Body metabolism and carbon dioxid output are increased, and changes in the total and differential leukocyte count occur—even when the light action is localized to one extremity. The pigmentation of the exposed skin is a protective measure, as pigmented skin is less pervious to ultraviolet rays. Some writers consider that only in albinos and those deficient in pigment is phototherapy with ultraviolet rays thoroughly effective.

Prophylaxis of Exposure to Ultraviolet Rays.—Industrial protection from these very active rays includes protection of both eyes and skin. To secure proper eye protection, work has been undertaken by Sir William Crookes and the German glass chemists. The technical paper No. 119 of the U. S. Bureau of Standards gives examples of such experiments carried on with different types of European and American glass to obtain maximum visibility with exclusion of violet rays. A condensed table from this paper is given:

PERCENTAGE TRANSMISSION OF WAVE-LENGTHS IN MILLIMICRONS

Glass	Ultraviolet Spectrum							Visible Spectrum						
	300	340	360	380	400	420	460	480	520	540	590	640	680	720
Crookes A	0	0	10	70	82	85	86	87	87	89	70	92	91	
Am. Opt. Co. 91B	0	0	10	68	80	83	84	86	87	89	66	92	91	
Am. Opt. Co. 57	0	0	30	46	75	84	89	90	92	92	91	90	90	
Am. Opt. Co. 58	0	0	0	20	50	71	73	75	80	82	84	84	85	
Noviol O	0	0	0	10	47	64	80	83	87	88	89	89	91	
Noviol A	0	0	0	0	0	10	74	80	87	88	87	88	88	

To protect the skin of travelers and others in high altitudes, various cosmetic preparations have been devised. Formerly burnt cork or soot was smeared on the skin. In industrial exposures, especially to the electric arc used in welding, more effective protection is required on account of the intensity of emitted rays and the nearness of the source—the effects of the rays decreasing proportional to the square of the distance. For skin protection there is a heavy mask with chest piece and gauntlets of leaded materials.

In general the eye-piece of the mask consists of several layers of glass, not only to prevent breakage but to cut off glare and heat. A common form consists of a red layer of glass between two blue layers; black, green, or yellowish green are also recommended.

Treatment.—The eye condition is extremely painful. Treatment is the ordinary treatment of conjunctivitis: bland washes, boric acid, or oil in the eye, and cool compresses. Instillation of cocain or atropin is often necessary for the severe pain. For the skin burn calamine lotion is most useful.

The lesions produced in the eyes by pure ultraviolet rays have been studied by the use of a special ray filter. This filter was employed during the war for a process of signaling between ships, where only the invisible ultraviolet rays from a powerful searchlight were transmitted and received on a fluorescent screen. Attempts have also been made to study the pathology from exposure to purely luminous sources—the violet rays being removed by color filter, and the heat rays by passage through liquids. This work has led to no definite results; and at present it is not known what structural damage may be caused by excessive light in the visible wave-lengths.

Eclipse Blindness

There is a form of ocular pathology called "eclipse blindness" which may be due to exposure to intense visible light. It results from looking at the sun with the naked eye, or through improperly smoked glasses, especially during an eclipse. The rôle of heat rays, and even of refractive errors, is still in question—although the condition has been known since Saint Yves in 1722 and much studied since.

The story and ophthalmoscopic picture are characteristic. After watching an eclipse (sometimes a sunset) with the naked eye or through a smoked glass, the patient notices that he can no longer see clearly. There is no pain, possibly photophobia, but he is particularly annoyed by a large diffuse floating cloud between himself and what he wishes to see. Later this cloud contracts and assumes a rotatory or vibratory motion. It may last for some months, and affects especially the near vision. Ophthalmoscopic examination shows changes in the color of the fovea, irregular pigmentation of the macula with punctate gray spots. There seems to be no close relation between the extent of the eye-ground changes and the defect of vision. In the majority of cases sight is slowly recovered, or at least the ocular defect is effectively compensated.

Under industrial conditions the same functional disturbance, although to a lesser degree, can be produced in those who are constantly looking into furnaces, or even into illuminating sources of great brilliance. Workers thus exposed should be adequately protected, since the defect of vision although not permanent is disabling, and may cause accidents as well as impair the quality of work produced.

Ocular Fatigue

In considering the visible rays apart from the others, it must be remembered that these are the only rays which fully reach the rods and cones of the retina. Heat rays are subject to the laws of absorption and refraction and only a few pass beyond the lens; the ultraviolet, even in the zone of 320 to 400 μμ, penetrate no further. The effect of visible rays is supposed to be a photochemical decomposition of the visual purple of the retina resulting in the sensation of light. When the eye is exposed to intense light, it is thought that the purple may be decomposed more rapidly than it is reformed; and that this exhaustion prevents the reception of certain color stimuli or even of light. The studies of Hecht on the decomposition rate of retinal purple by light of various wavelengths and intensities offer some support for these assumptions. But whatever the theoretical aspects of the question, subjective symptoms of ocular fatigue and even serious damage, from the rapid reception by the retina of stimuli of intense brilliance, are a grave and very real industrial problem.

Although little is known about the adaptive processes of the retina, adaptation by the voluntary and involuntary musculature of the eye has been more thoroughly studied. It is possible to understand how ocular fatigue may arise from undue strain or conflict in these mechanisms. The majority of eye-strain is due rather to ill-placed or insufficient illumination rather than excessive lighting; but there is a well-recognized fatigue from glare, or from exposure of the eye to what illuminating engineers call sources of high intrinsic brilliancy. Such a source may be a naked flame, an arc, the filament of an electric bulb, or the sun; or it may be the reflection of such source from a shining or polished surface, part of a machine, a book page, or a mirror. In other cases where the emitted light does not in itself seem excessive, fatigue occurs in an eye adapted to a condition of lower illumination. No adequate definition has been made of what constitutes glare and at present there is no clinical differentiation between the various forms of ocular fatigue—except that in cases of intense light exposure there may be retinal changes, color scotomata, and even blind spots as previously mentioned.

In the fatigued eye there is hyperemia, even inflammation of the conjunctiva. The eyes feel hot, dry, and itchy; there is increased frequency of winking, and a desire to rub the eyes, which temporarily relieves. Attempts have been made to count the rate of winking as an index of fatigue. Fatigue of the muscular adjustment expresses itself by blurring of vision, difficulty in keeping the eyes open and sensitiveness to light. The more remote symptoms are pain in the eyes, the orbit, or headache. The eyes have been called the most "psychic" of organs and eye-strain has been the cause of functional disorders in many parts of the body, such as neuralgia, lassitude, nausea and indigestion.

Prophylaxis.—The causes of eye fatigue from faulty illumination are dim light, shadows on the working surface, lack of necessary contrast, flickering or unsteady sources of light. Although the mechanism of the production of fatigue is still theoretical, there are certain simple facts.

If the light is dim, the pupil will dilate; yet if the attention is fixed at the same time, the pupil attempts to contract to shut out disturbing peripheral rays. Shadows that fall across the work interfere with the adaptation of the eye; if adapted to high light, shadow vision will be dim; if adapted for shadow, the high light will be excessive. On the other hand, in certain types of work, shadows may be needed for contrast. Flickering or moving light is the cause of similar conflict, since there will be fixation of attention with a constant attempt to change the mechanism of adaptation. It is such conflicts that result in strain; and, where the adaptive mechanism is already burdened by refractive errors, the strain is twofold.

Miners' Nystagmus

There is a special form of industrial eye fatigue due to poor illumination of great economic importance in European countries. This is *miners' nystagmus.* It is found in underground miners, and is characterized by rapid symmetrical circular or horizontal oscillations of the eyeballs at a rate of from 100 to 300 times a minute. Oscillations are more marked on looking upward; they are synchronous, persistent, and aggravated by darkness. There are also photophobia, lid-spasm, failure of visual fixation, head tremors, and often marked psychoneurotic symptoms. In mild cases the subjects are unaware of the oscillations, but in severe cases the victims are practically helpless and are led about by their wives or children. The miner becomes aware first of inability to see clearly, then to direct his pick accurately or to find his belongings. Lights and objects begin to dance about him, and he is at last obliged to abandon his work. Usually after a certain length of time aboveground the symptoms disappear, and the man is able to return to underground work. If this is under the same conditions relapse is prompt to follow.

The disease was first described by Decondé in 1861. Most of the early studies were made in the north of France and Belgium, especially by Dransart and Romiée, who, however, held opposite views as to its causation. Valuable modern studies have been made: in Germany by J. Ohm, who has done work on the miners of East Prussia and devised ingenious apparatus for recording and measuring the ocular movements; in Belgium by Stassen; and in England by Llewellyn, who first actually determined the feeble illumination of the coal face. This recent work is summarized in the British Medical Research Council's Report of the Nystagmus Committee, which contains an extensive bibliography. In European countries the disease is found only in certain localities, but where found seems to affect from 20 to 25 per cent of the miners. In England there has been of late a marked increase in the number of reported cases on account of the compensation law and industrial conditions. In America the disease seems not to be known. Hoffmann for the

U. S. Bureau of Mines attempted a statistical study but was unable to find records of any cases and was obliged to fall back on the assumption that there should be "a possible, if not probable," estimated number of cases. A number of factors may combine to account for the infrequency in the United States: (1) Nystagmus is not a compensable or reportable disease in many of the coal-mining states. (2) American working conditions are different. For example, in the unionized Central Competitive Field—Ohio, Indiana and Illinois—the mines are usually not deeper than 250 feet, they are relatively dry, the workers come and go freely and are able to be at outdoor work around their homes in daylight. They work as a rule only part of the year and spend the rest of their time on farms and at outdoor work. The seams worked are never less than 4 feet in thickness (usually 6 feet or over) and electric lighting is more common—although at the working face the safety lamp is still used.

In the causation of nystagmus, many factors have been accused, such as visual monotony, refractive errors, posture, fatigue from looking upward, poor lighting, glare from open lights, glare and unequal reflection from coal surfaces, poisoning by carbon monoxid, methane, or alcohol, and atmospheric pressure, heat, vitiated or humid air. In about 10 per cent of all the cases there has been an etiological factor in the form of accident, injury or disease. It is assumed that the symptoms were already present but only under these circumstances were they brought to the attention of the patient.

The Report of the British Commission, after consideration of the various factors, gives as its opinion:

"1. The essential factor in the production of miners' nystagmus is deficient illumination. Other factors, such as position during work, accident, alcoholism, infections, malnutrition, hereditary predisposition, and errors of refraction are of secondary importance only, while depth of workings, thickness of seams, and the ordinary gaseous impurities in mine air have no direct influence on the disease.

"2. The deficient illumination is due to the low illuminating power of the safety lamps generally used by coal miners, to the distances at which these lamps have to be placed from the objects which the miner has to look at, and to the great absorption of light by the coal and the coal-dust-covered surfaces. In addition the effect of coal dust or dirt in obscuring the lamp glasses, the choking of wire gauze chimneys and the presence of moisture or low oxygen percentage in mine air, all reduce the light given by oil lamps, while failing voltage, poor bulbs, or lack of proper attention have similar effects on the illumination given by electric lamps.

"3. Workers at the coal face are more affected than other underground workers, and this appears to be due to the unrelieved blackness of the coal and the greater need for accurate vision.

"4. Distinct signs of nystagmus are present in a large proportion of coal miners, though only in a small proportion do the symptoms ever become so severe as to cause even temporary incapacity for work underground."

A serious complication is pronounced psychoneurosis, due in some cases to the ordinary domestic, sex and other non-occupational causes, but in others clearly due to the loss of self-confidence and judgment caused by the uncertain vision, or even to fear from working under dangerous conditions with failing eyesight. Other factors are the supposed effect of some accident in the causation of the disease and the effect of compensation or fear of loss of vision.

The nystagmus is usually obvious enough, but in doubtful cases can be elicited by requiring the patient to look upward while his head is bent forward. This will produce nystagmus or a spastic internal strabismus, and often a tremor of the head. Usually there is lid-spasm and photophobia, and the affected miner wears his cap drawn down over his eyes. Day-blindness (hemeralopia) has been reported by Weekers of Belgium. Shyness, stupidity and other symptoms in the psychic sphere are not uncommon in the psychoneurotic.

Treatment.—Except in the cases with severe neurosis, cure is certain if the worker is kept aboveground. The color of green fields is considered to be helpful. Compensation plays a great part, and on this account certain English writers believe that it should be limited to two years, or that settlement for a lump sum would be desirable as men could then be induced to return to work. More curative perhaps is part-time occupation while compensation is continued to make up a portion of the difference in wages. In spite of any palliative measures, the cost in compensation for this disease for the year 1920 in England was £300,000—an expense which will some day be obviated to a great extent by proper lighting. Portable electric cap lights where possible offer a steady form of light of fair intensity, although where there is concussion from blasting they may not be utilizable. In such case naked flame lamps are preferable, as statistics from English and Belgian sources show much lower incidence of the disease where these lamps are used.

Eye-strain and Poor Lighting

There have been numerous attempts to estimate the amount of eyestrain in an ordinary industrial community. Some arbitrary figures have been given of the amount of hazard incurred by various groups of workers. Gould estimates that about 40 per cent of all persons working are subject to conditions producing eye-strain. He discusses also the rôle of errors of refraction in producing eye fatigue, as well as the production of the refractive errors from poor lighting and eye fatigue—referring especially to the myopia of school children. More recently an estimate has been made from another direction, in the form of a survey made under the direction of the National Lamp Works reported by Eastman to the Society of Illuminating Engineers. They investigated the condition of lighting in 446 industrial plants and factories and classified their findings as follows:

	Per Cent
Excellent	8.7
Good	32.0
Fair	29.1
Poor	18.8
Very poor	3.5
Part good, part poor	7.8

They emphasize that the loss to the employer from poor lighting, and which he pays for in spoilt work, in accidents, in poor health and morale of his employees, is not properly appreciated.

Studies such as the above and the code of the Illuminating Engineering Society, which is the base of such state laws as are now in force, have been made possible through the recent perfection of an instrument called the photometer. This measures by comparison with standards the amount of illumination on any surface in units known as foot candles. A foot candle is the intensity of illumination on a surface lying at a distance of one foot from a one-candle-power source of light.

By the use of this or similar instruments in working out the problems of natural (daylight) illumination, tables have been made of the seasonal and daily variations of light indoors and outdoors, of the amount of light available from daylight at different times of the year, and other matters of importance to the illuminating engineer. Objects outdoors affect the amount of light entering, as does the character of the window glass, clean or dirty, plain, frosted, prismatic or colored. The hygienic value of sunlight must be considered, but the direct light should seldom reflect from working surfaces. The use of transparent panes in the lower part of the window prevents monotony by allowing the worker to look away from work and at other colored objects. Finally north light, being uniform in quality, is often desirable and can be obtained in one-story buildings by a "saw-tooth" roof.

In considering artificial lighting, intensity, steadiness, shadows, and absence of glare are important. The quality of the light is much less so. Verhoeff and Bell have shown that no artificial source of light used for illuminating purposes contains enough ultraviolet radiation to be injurious

FIG. 4.—READING THE FOOT CANDLE METER. (Courtesy National Lamp Works, General Electric Company.)

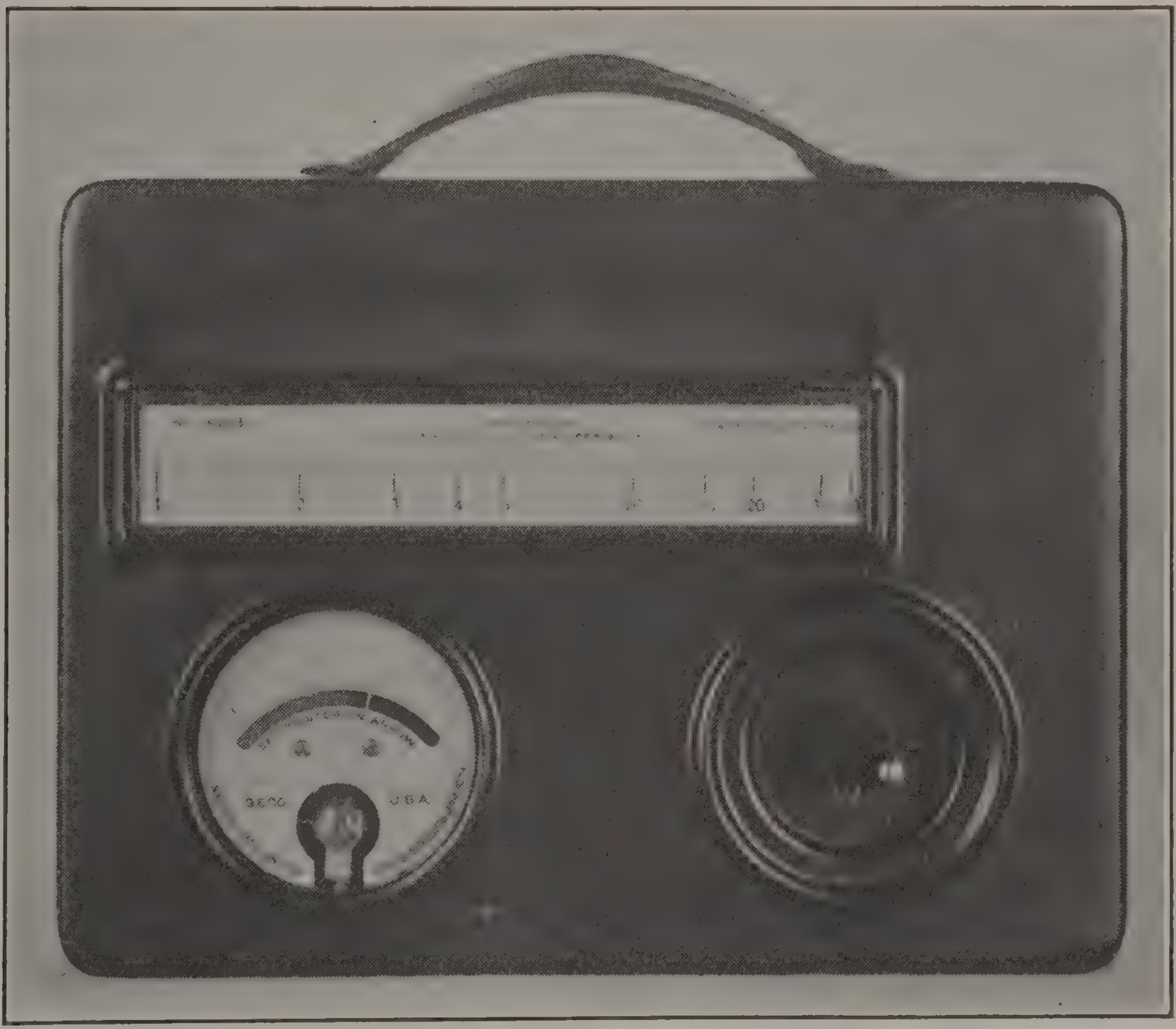


FIG. 5.—FOOT CANDLE METER. (Courtesy of the National Lamp Works of the General Electric Company.)

to the eye under practical working conditions, while the infra-red rays have no specific action distinct from thermal effect. Some prefer kerosene or gaslight to the electric light on account of the relative absence of blue or violet rays; but the mercury vapor lamp is successfully used in drafting rooms. In considering non-electrical lighting, account must be taken of the products of combustion, heat, and noxious gases. The general types of lighting are direct, semidirect, and indirect. Direct lighting means light from an electric bulb with visible filament, or from an open flame. This must be shielded from, or well above, the level of the worker's eyes to avoid glare. In semidirect lighting the glowing source is covered by frosted glass or other semi-opaque medium. The most agreeable light is from indirect lighting. Here the source is not visible, but the reflection from a white wall or other object is utilized to diffuse the illumination. Each of these kinds of lighting has specific advantages. Furthermore, light absorption by wall color and other objects must be considered.

Prophylaxis.—The above are some of the problems of the highly developed science of illumination—some of whose followers feel that their best results will only be obtained when they can exclude the uncertainty of nature, and light all work from purely artificial sources. In any case the prevention of industrial eye-strain is essentially a problem of the illuminating engineer and is only to be attained by installation of lighting adequate to the needs of the work. It is well for the physician to know enough of this subject to recognize faulty illumination, and to realize the symptoms of eye fatigue in those exposed to its effects, although some would limit his usefulness to the recognition and adjustment of errors of refraction—errors which if uncompensated mightily increase the burden of the strain.

RADIATION

Roentgen rays were discovered in December, 1895; and, although erythema, ulceration, and loss of hair were noticed in the next year, it was long before the extent of the chronic skin changes was realized. Finally in 1902 the first case of skin cancer in a radiologist was reported. Porter, in 1909, collected 39 cases of cancer, on 13 of which he had operated personally. Up to 1911, 67 cases had been noted.

In their ignorance of the delayed but serious results of the action of the rays, the early experimenters exposed themselves recklessly, testing out the tubes with their hands and demonstrating the presence of the rays by fluoroscopy on their own hands and persons. Besides, the apparatus at that time was variable and uncertain and required exposure of the operator for adjustment and control. The immediate effects were not felt, and the development of skin lesions was so slow that the damage was well under way before it was thought of. In any case, so little was then known of the nature of the rays that complete and intelligent protection was not possible.

The nature of the X-rays is similar to light. Visible light is the result of electromagnetic waves of a length from 0.00076 to 0.00038 mm., oscillating with a rapidity of 400,000,000,000 to 800,000,000,000 times a second. X-rays are shorter and of a proportionately greater rate of vibration, since the speed of all these electromagnetic vibrations is the same.

Wave-length in Millimeters	
42-36 $\times 10^{-5}$	Violet light
10 $\times 10^{-5}$	Ultraviolet rays
0.003-0.005 $\times 10^{-5}$	Roentgen rays
0.005-0.0001 $\times 10^{-5}$	Radium rays (gamma)

X-rays analogous to light have many different wave-lengths and form a spectrum of their own which can be analyzed by means of crystal planes (Bragg, Moseley). The magnitude of the waves is less than .0000001 mm. and the rapidity of vibration more than 300,000,000,000,000 times a second. The displacement of the vibrations is in the realm of atomic measurements.

Under the influence of the recent knowledge of radium and X-ray measurements, the atomic theory has been expanded into a rather well-rounded logical whole. Matter, in its smallest homogeneous particles, consists of molecules. These molecules are formed of smaller structural subdivisions called atoms, of the same or different kinds. They are held together by electromagnetic forces and are incapable of existing alone except in some sort of electrical equilibrium. Atoms are pictured as small solar systems, consisting of central positively charged nuclei surrounded by a number of rapidly moving negatively charged units, the sum of whose charge is balanced by the total positive charge on the nuclei. These vibratory satellites each carry a single unit of negative electricity and are called electrons. Their number and speed of rotation determine the chemical activity of the atom. By certain physical forces electrons can be torn away from their nuclei and may travel for a short time as independently charged little bodies, until attracted by or repelled into some other atomic system. The eruption of electrons from their system or their impact with new atomic systems produces minute electromagnetic disturbances of the nature of light, which are the X-rays or the gamma rays of radium.

X-rays are produced for practical purposes in two ways: First through a modification of the original Crookes' tube. This is a vacuum tube through which is passed a current of electricity under high tension. The passage of electricity means the passage of negatively charged particles or electrons from the negative pole (cathode) to the opposite pole or anticathode (anode). There is also a positive discharge towards the cathode, the nature of which (so-called "Canal Rays") is not clear. Electrons from the cathode travel with a speed of 10^9 to 10^{10} cm. a second (about one-third to one-thirtieth of the speed of light) varying with the tension of the discharge; and where they strike on the opposite pole X-rays are given off.

The second practical method is the Coolidge tube. Here use is made of the principle that metals (tungsten), heated white-hot by the passage of electricity, give off electrons in number proportional to the temperature. At the time of impact of these on the anticathode, X-rays are given off. This apparatus is almost universally used in America, and offers advantages in control and steady adjustment.

The waves spread out in the form of a sphere or cone from their focal point. Their effect decreases markedly with the distance, their intensity diminishing proportionally to the square of the distance. Besides the distance from the source, the intensity of the rays varies with the current passing (milliamperage) and with the voltage. The voltage determines entirely the character of the rays—that is, their wave-length. On this depends the penetrating power. If the voltage is low ("soft tube"), the X-rays are of long wave-length. These are absorbed or transformed in the skin and superficial tissues. It is the short waves from the high voltage tubes ("hard tubes") with their rapid vibration that penetrate deeply.

The action of various types of X-ray is not thought to differ except in penetration; but where absorbed, the effects of equal doses (the product of time by intensity) on cellular pathology is the same. The effect is due to the total quantity of rays and is the same whether the quantity is delivered rapidly or slowly—and whether the waves are long or short, from high voltage tubes or filtered through metal screens.

According to the present ideas of treatment it is only the shorter waves which are desired. They penetrate deeper and are more nearly of the same length. The long waves are uncertain, easily deflected, and cause skin burns and secondary troublesome effects. Although the amount of short rays reaching the deeper tissues is necessarily small, since even these will burn the skin if in sufficient amount, nevertheless, by "cross fire" radiation from different directions, effective dosages at various depths can often be obtained. The ideal is to obtain homogeneous rays approximating a single wave-length. In this the short waves produced by high voltages are valuable, since they are of great intensity in but a single zone characteristic for each voltage, until a voltage is reached at which the characteristic secondary rays of the target itself are given off

(80,000 in the case of tungsten). It is calculated that a tension of about a million volts would be required to produce waves as short as the gamma rays of radium. This voltage has been attained experimentally, but the highest in actual practice is about 200,000.

The gamma rays of radium are one of three kinds of "rays" given off by radium. Radium is a mineral in a state of molecular decomposition. Atoms are disintegrating and given off from the molecule until eventually the residuum is a new chemical individual. In the case of radium it is a gas, radium emanation, which itself is undergoing radio-active destruction. A whole series of transformations has been studied by the discoverers of radium and by the English physicists, Rutherford and Soddy. The largest and most powerful rays from radium or its transformations are the so-called alpha rays. These are not rays but are the heavily charged electropositive atomic nuclei projected from the molecule at extremely rapid speed. These relatively large masses at high speed, when passing near other atoms, can cause dissociation or other molecular rearrangements by setting electrons free. A second type of radium rays, the beta rays, are negative electrons from the atoms, liberated at various rates of speed; although all those from radium are relatively rapid, with a speed about ten times that of electrons in the vacuum tube. These electrons, as well as those liberated by the alpha rays, by impact or other changes in velocity, give rise to very fine electromagnetic vibrations of extremely short wave-length, since the speed of the electrons is so rapid. These are the gamma rays.

Besides the rays actually given off from the X-ray tube and radium, there are several kinds of secondary rays produced in tissues or other substances under the influence of radiation. Most important are the so-called secondary characteristic rays. When X-rays or gamma rays penetrate a foreign molecule, they may liberate electrons and electromagnetic waves, which in the case of the heavy metals are of definite character for each metal and depend on the atomic weight of the molecule attacked. Those from other substances than the heavy metals are not so well studied, although they are known to be formed from bones and tissues as well as the lighter metals. X-rays or gamma rays can also be reflected from atomic reflecting surfaces as any of the other forms of electromagnetic waves. Recent construction has removed many of the secondary rays originating in the apparatus, but those arising in the tissues are not so easily controlled. Distance from the source scatters the long and erratic waves. Metal filters and screens, whose secondary rays must be guarded against, can be used to make the type of ray reaching the tissues more uniform and thus allow some quantitative estimation of the secondary effects.

The action of the two substances, radium and X-rays, can thus be understood to be the same. In X-rays it is only the electromagnetic oscillations formed by the impact of electrons on the anticathode that reach the tissues.

Radium.—In the case of radium, the actual atomic fragments in the form of alpha and beta rays may reach the superficial tissues. The penetrating power of both alpha and beta rays is slight. At a distance of 1 cm. the alpha radiation is effective to a depth of only 0.058 mm. While at a similar distance beta rays penetrate to 1.5 cm., gamma rays and X-rays penetrate deeply. It is believed that the effect of the alpha and beta rays (which are not "rays" at all), as well as that of the electromagnetic rays, depends on their power to produce ions in the tissues which absorb them. In the case of electromagnetic rays the ionization may be caused by production of electrons in the tissues. In any case the effectiveness of the radiation at any depth is best measured in terms of its ability to produce ions.[7]

The modern method of estimating intensity of radiation for control or dosage is the measurement of ionization of gases. Gases that do not readily conduct electricity may, under the influence of radiation, become fairly good conductors. This is due to separation of gas molecules into charged conducting particles (ions), and this ion formation is proportional to the intensity of the radiation. This conductivity is determined on the surface or by placing the instrument at various depths below water, since the absorbing power of the body tissues for radiation is about that of water.

The sensitiveness of the different tissues is said to be as follows: Most sensitive are actively dividing cells, the cells of the graafian follicles, the testicular epithelium and the cells of rapidly growing tumors. Cells of simple protoplasm and nucleus are more readily affected than highly differentiated elements—although the more delicate function of the latter is easily affected. Dehydrated cells and tissues, and such generally resistant structures as fibrous connective tissue, are highly resistant to X-rays and radium. Although cytoplasm as well as nucleus is affected, the most delicate changes noted are a graded series of effects on cell function. First, the germinal cells lose their ability to transmit the finer species differentials and hereditary factors; then defects occur in the delicate highly differentiated organs such as brain and eye. Eventually differentiation of simpler tissues is lost. In individual cells the first function to be impaired is motor activity and cell reproduction, next oxidative processes, and finally the visible structure and the life of the cell itself. The action is graded according to the dosage. Lately there has been an attempt to use light doses for stimulating effect, although the original basis of therapy was that of selective destruction. There may be a summative effect between unfavorable tissue conditions such as injury or infection, which leads to increased radiation effect.

---

[7] The total number of ions produced by the various rays and their penetrating power is about as follows:

1 mg. radium α rays at distance of 1 cm. from surface 1 $\overline{\text{cm.}^2}$ produces 1.67 by 10^{12} ions; all in layer 0.058 mm. in depth.

1 mg. radium β rays at distance of 1 cm. from surface 1 $\overline{\text{cm.}^2}$ produces 7.5 by 10^{10} ions; all in layer 1.5 cm. in depth.

1 mg. radium γ rays at distance of 1 cm. from surface 1 $\overline{\text{cm.}^2}$ produces 1.1 by 10^{11} ions; at depth of 12.5 cm., 50 per cent of ions have been formed; at 41.0 cm., 90 per cent; and 82.5 cm., 99 per cent.

X-rays, 10 milliamperes, 90,000 volts, 0.2 per cent efficiency.

Total ions—1.5 by 10^{14} with surface at 20 cm. from tube at a distance 40 cm. below surface only 89 per cent of ions have been formed.

The effect on the germ cells of workers in X-rays and radium and the resultant sterility has long been known. In accordance with the statement above it has been shown that the tubules and ovarian follicles but not the interstitial cells are affected, and that the growth curve of animals exposed was normal. Experimental production of monsters from radiated embryos has been observed; also the gradual evolution of defects was noted in the later generations of animals, radiated without effects on the individual of the first succeeding generation.

Although radium allows the physical contact of atomic particles with the skin as well as electromagnetic rays, and although the gamma rays are shorter and more rapid than X-rays, it can be understood, since only the ionizing power is effective, that the action of radium differs merely quantitatively from the X-rays. There are, however, certain clinical differences due to the differences in producing and handling the two forms of energy; and in general the radium effect is quicker in developing, produces more inflammatory reaction and subsides more quickly. This is possible because, although very powerful and in intimate contact with the tissues, the amounts used by the very nature of the substance must be small.

Radium is usually used in the form of emanation. This is the gas which is the first transformation product of radium and contains especially active alpha and beta particles. It is drawn off by air pump into small capsules of glass or metal whose activity, and hence whose usefulness in dosage, can be determined. These applicators are placed in intimate contact with tissues to produce alpha and beta ray effects. To obtain the gamma ray effects, radium itself is used screened by lead sheets which absorb the alpha and beta rays but allow passage of the gamma rays into deep tissues. Workers pumping off the emanation, although protected by screens in other ways, must expose their hands, as the work is too delicate to permit of heavy gloves. Nurses and physicians making the applications are under a similar disadvantage, although forceps have been introduced which increase the distance between the hand and the radiating source and obviate in great part the effect of the alpha and beta rays.

The clinical picture of occupational radium dermatitis as described by Ordway is usually a lesion of the radial surface of the first two fingers and of the ulnar surface of the thumb. These are the surfaces exposed in handling the applicators. Several days to weeks after careless handling, pain and sensitiveness develop in the fingers. The skin is red, slightly swollen and shows marked sensitiveness to heat and cold and loss of fine tactile sense. This is persistent and often interferes with work. In addition there may be boggy subcutaneous swelling that pits on pressure and only slowly returns to its original shape. The nails are cracked, brittle, and stand away from the flesh. In the course of weeks desquamation occurs, but the tenderness and paresthesia only very gradually disappear. If the worker continues careless, chronic skin atrophy, intractable ulcers, hyperkeratoses and even cancerous degenerations occur—as in the typical "X-ray hand."

Dermatitis.—X-ray dermatitis, usually called "burn" and produced as a result of treatment, may occur in any part of the body sufficiently radiated. Burns are usually classified as first, second, or third degree. In addition there is often an immediate systemic reaction or intoxication which seems to result from local tissue destruction, since it can be prevented by obstructing the circulatory return from a radiated member. Although such burns are never occupational, but rather incidental to a course of treatment, they are worth describing. The first degree burn is a faint or angry erythema, developing in one to two days to three weeks, whose severity is determined by the rapidity of its production. It is mild and of short duration; usually the hair falls out, and, if the reaction is severe, does not return because of atrophy of the hair follicles. There is itching, burning or stinging. The blush may first be noticed when bathing the affected surface; it is said to be more readily visible by mercury vapor light. The second degree burn shows erythema early. Following this comes edema and vesiculation. The outer layers of the skin desquamate, leaving the papillary layers of the derma exposed. This process lasts several weeks to months. Healing is spontaneous. Hair is permanently lost. In severe cases the pain is intense. In the third degree burn the early erythema is followed by a beefy redness of the tissues and deep edema. In a few weeks dry necrosis occurs. Where the necrosed area sloughs there is a purulent discharge; the border shows inflammatory thickening. Healing requires many months, or else an indolent ulcer persists, which is punched out and well defined, with an indurated margin and a floor dry and glistening or covered with unhealthy granulations. The adjoining skin may show atrophy or telangiectasis. Subjective symptoms are severe, almost intolerable pain, burning and aching. This pain may cease with the sloughing or persist until healing, but the chronic ulcers are painless. After effects of all burns in the form of pigmentation, atrophy of the skin, telangiectasis, and thickening of the horny layer are common. The severer reactions result in scars, fissures, sclerodermatous or keratotic degeneration with surrounding areas of pigment, atrophy, or telangiectasis.

It is this profound chronic dermatitis, especially of the hands, which characterizes the professional radiodermatitis. With patchy pigmentation and atrophy, the skin is smooth and glistening. Besides atrophy the hand shows fissures and ulcers with desquamation and hyperkeratosis. This type of skin is prone to cancerous degeneration. The cancer described by Wolbach is an ordinary "prickle cell" cancer without peculiarities except its occurrence in comparatively young persons. It develops slowly and spreads by the customary lymph channels to epitrochlear and axillary glands, whence, unless prevented, it makes its way into the mediastinum and the general system.

The location of the dermatitis is usually the dorsum of the hands, since the majority of cases occur in those who used their hands in palpation or in testing their tubes for photography or fluoroscopy. It seems that ordinary clothes protect from the changes (which are thought to be due to the long waves) since the skin effect ceases almost invariably at the cuff-line. In technicians engaged in the manufacture of X-ray apparatus, lesions of the face are common; and in habitual subjects for fluoroscopic demonstration, pigmentation, keratoses, and cancer have been found on the skin of the chest.

The histological picture shows occlusion of the blood-vessels of the derma, caused by proliferation of the endothelial walls. This results in atrophy, exudation, telangiectasia. Nerve endings caught in the proliferating tissue are thought to be the cause of pain. There is atrophy of the integument and hyperkeratosis; sometimes the horny layer extends downward and invades the derma. It is in the cases of chronic ulcers or in the hyperkeratoses that malignant degeneration occurs.

In addition to the skin changes, cases of profound anemia without signs of blood regeneration (aplastic anemia) have been reported in radium and X-ray workers, from which there has resulted much speculation in regard to the effects of radio-active rays on the blood-forming organs. That workers in X-rays show certain marked characteristic blood changes was first remarked by Gudzent and Halberstaeder. They showed that leukopenia with an actual lymphocytosis existed, along with a slight diminution in the red coloring matter. The cause of this blood picture and of the reported occupational anemias has been attributed to the action of radio-active substances on the blood-forming organs, to poisonous gases or emanation in the laboratories, to fatigue, lack of fresh air or exercise, and to strain. Gases such as ozone or the various oxids of nitrogen are found in the proximity of X-ray tubes; the emanation of radium is in the form of a gas, and the ordinary air constituents are in an ionized state due to radio-activity.

Prophylaxis.—The subject of the toxic properties of the gases has been studied seriously in this country and Great Britain, and the Report of the British Committee recommends the following measures as preventive:

> "General Recommendation.
>
> "1. Not more than seven working hours a day.
>
> "2. Sundays and two half-days off duty each week, to be spent, if possible, out of doors.
>
> "3. An annual holiday of one month or two separate fortnights.
>
> "Nurses and whole time workers in X-ray and radium departments should not be called upon for any other hospital service."

In addition to providing for protective screening of the operator with at least 2 mm. of lead for superficial therapy, and 3 mm. of lead for deep therapy X-rays, they insist upon the importance of airy, well-ventilated rooms, which can be sunned from time to time.

For radium they recommend inclosure within at least 1 cm. of lead for handling, and forceps for manipulation. Where radium is stored it should be inclosed in a thickness of at least 8 cm. of lead; and operators should not stay in its vicinity longer than necessary.

Emanation should be handled in an inactive state as far as possible. If handling is necessary, thin rubber gloves should be worn. Escape of emanation into the air should be guarded against, and the room should be fitted with an exhaust fan.

Treatment.—The treatment of the dermatitis varies with the extent of the changes and atrophy. Above all to be stressed is the avoidance of any skin irritants such as alcohol, ether, formalin, glycerin or bichlorid. In the earliest stages a simple powder is best. Dry cold and cooling lotions used cautiously often serve to abort the erythema. Lanolin and whitewash are excellent. In the chronic ulcers of the third degree, complete excision of the ulcer and damaged tissue, when possible, hastens healing. If this is done before complete delimitation of the lesion, there may be sloughing and delayed healing. Lately in Europe treatment has been offered in the form of beta rays of radium used to point of a reaction, or else its equivalent in the soft tube X-rays. The keratoses have been treated by the application of carbon dioxid snow, or beta radium. A single local cancer is said to respond readily to the action of beta radium. At present the effect of these soft radiations on the already atrophic skin is still a subject of discussion. Surgical treatment in the hands of the experienced operator seems to offer the best hope of a permanent cure.

The skin of the radiologist once affected is extremely sensitive to small doses of X-rays and ordinary protection is no longer sufficient, since secondary rays of ordinarily harmless intensity, or even spent X-rays which pass through a thick lead screen, may add their quota to the dosage he receives. Owing to the present use of increasingly high voltages this matter is of great importance, and Pfahler recommends that operators carry dental films in their pockets which should be developed every week to show the amount of this exposure. The protective devices are technical construction problems, and consist of lead screens, leaded glass windows for observation, and control checks, making it impossible for any one not enclosed by protective screens to use the apparatus. Even with gloves the fluoroscope presents a certain hazard to the affected skin. At least temporary cessation or alternation of occupation is advisable in all cases; although with the steadily increasing knowledge of pathological effects and of preventive measures, it is becoming clear that the future will not require the lives of so many martyrs to investigative zeal.

ELECTRICITY

At present the most widespread use of electricity is for light and power, and the workers most exposed to electrical occupational injuries are the men employed in power houses where electricity is produced, and on the lines transmitting it to its ultimate use in streets, buildings and railways. There are many industrial uses besides those of light and power. The heat from resistance to electric currents is employed to produce the high temperatures of the electric furnace; the heat of the spark gap is used in welding and attains several thousand degrees Centigrade; besides this, there is a whole modern field of currents of high voltage, usually, to be sure, of low intensity, which has grown up in wireless and X-ray work. Workers in all these fields and those engaged in the manufacture of electrical apparatus may be especially exposed to hazards of electrical injury.

Electricity is commonly compared to a fluid driven through a fixed channel and exerting a force dependent upon the pressure behind it. If the transmitting wire is considered the pipe containing this mysterious fluid, the pressure behind is called the voltage; the amount of electricity passing the amperage, and the obstruction offered to passage, are the resistance. The relation of these three properties to each other is stated in Ohm's law. $\text{Amperage} = \frac{\text{Voltage}}{\text{Resistance (Ohms)}}$ expressed in appropriate gram centimeter second units.

There are two kinds of electrical currents in general use, the alternating and the direct current. The direct current is a continuous uniform flow from one pole of a generator to the other, or between two points of different potential; while the alternating current is a current whose direction reverses constantly and whose intensity is varying between alternating maxima in opposite directions, and is diagrammatically expressed as a series of waves. Ordinary lighting currents have about sixty cycles a second, while some of the high tension currents used in scientific work may reach several thousand. In considering the industrial hazard of electricity, it is probably the tension which is of greatest importance, since it is this property which overcomes the opposed resistance of skin, tissues, or other insulators, and allows the passage of current according to Ohm's law. Yet many high tension, high frequency alternating currents are of such feeble intensity that they can be handled as harmless and pass through the body in various forms of medical electrical treatments.

The ordinary dry skin resistance of the body, for instance of the hands, is very high although the actual measurements reported vary. Through soft moist parts of the body surface, conductivity is much greater and may equal that of the tissues, approximately the conductivity of normal salt solution. In the same way such outer factors affect the susceptibility of the skin as wet or dry contact, earth, rubber shoes or gloves, clothes, or boot nails. This makes it difficult to estimate the danger of any particular voltage. Besides the voltage and skin resistance, other factors concerned in the production of damage are the intensity of the current, the duration of the contact, and the vitality of the body.

The kinds of injury caused by electricity are burns, mild or fatal shock, and ophthalmia. Any or all of these may result from the interposition of a part of the person between two conducting bodies of different tensions, or else by the grounding of a circuit through contact. Sometimes in the neighborhood of high tension wires a spark will discharge upon some relatively remote person or object that is grounded.

Electrical Burns

Where sparks or contacts with high tension occur, there invariably results a burn. At times the burn is small. The burns are found at the points of entry and exit of the current from the body. Large flaring sparks char the skin and coagulate the tissues through the effects of heat, while the passage of the current in itself produces electrolysis in the neighborhood of the burn. This makes healing very slow. Coagulated tissue proteins do not recover; the electrolyzed tissue recovers slowly and only partially. Apparently small burns may slough and necrose away to the bone, involving grave danger of secondary hemorrhage. A characteristic of badly burned flesh may be deposits, thought to be flakes of stearin, in hard white cartilaginous-appearing plaques. At times the subjacent tissues are devitalized and undergo necrosis where the skin itself is apparently intact. The burns are slow to heal, often painless, and, unless subsequently infected, not inclined to suppurate.

The histological picture of burns outside of the charred area has been described as a spindle-shaped arrangement of cells of the rete malpighii, and of smooth muscle fibers in bundles parallel to the line of action of the current.

The burns produced by direct currents are in general severe. Alternating currents, especially of high frequency, prevent much of the electrolysis in the tissues by their rapid reversal. Heat, however, is formed during the passage of the current, and this property is used in treatments (diathermy). Burns of skin from alternating currents may be severe, depending on the spark and the length of time of its action. More often the portal of entrance and exit is small and may be found only after some search. The skin action of direct currents of any tension is usually more severe and the severity increases with the time of action, as electrolysis continues.

Treatment.—The treatment of electrical burns is slow. There is much sloughing, and the extent of the lesion, although it is always greater than it appears, cannot be accurately foretold often for a long time. Dry treatment, sterile petrolatum, or one of the new paraffin sprays are the most valuable. Wet dressings may be used in infected cases. The constant danger of secondary hemorrhage must be borne in mind.

Electrical Shock

In regard to electrical shock the question is one of degree. The clinical picture produced, besides burns at the portal of entry and exit, is of tonic spasm and rigidity of the muscles, including the respiratory muscles, during the passage of the current, heart fibrillation, paralysis, and failure. The subject falls with a peculiar cry due possibly to muscular rigidity. Often when the leg muscles are involved, the victim leaps or is apparently thrown a considerable distance. At death the heart is full of blood in fluid condition; there are minute punctate hemorrhages in the heart and central nervous system; with excessive currents, the blood is hemolyzed.

The cause of death from passage of electric currents has been much studied since the first experimental work of Prevost and Batteli. There has been much discussion as to whether respiratory paralysis due to action on the medulla occurs, or whether death is due to cardiac fibrillation—delirium cordis. It is known that respiration will cease while heart action continues, but this is considered due to tonic spasm of the respiratory muscles which ceases the minute electrical contact is broken. At other times respiration continues after the heart has ceased or gone into fibrillation. The heart action, if suspended, may resume spontaneously, but this never occurs after two minutes.

The characteristic effects of various tensions and their safety limits have also been studied. The direct current (although fatalities in subjects well grounded have been reported as low as 95 volts) is considered reasonably safe at 300 volts. At lower voltage shock may be severe and cause fainting and collapse, especially in emotional subjects and those of low resistance. With increasing voltage of direct current, the danger increases. Oliver, however, mentions an electrician who handled several thousand volts. Alternating currents in slow cycles (12 to 75 a second) are dangerous even at 110 volts. They increase in safety with rapidity of alternation and especially at high voltages. With direct current, cessation of the heart occurs at lower voltages but leaves the respiration unaffected. At higher voltages the muscles are drawn into opisthotonos. With alternating current at low voltage, cardiac fibrillation readily occurs. It must be emphasized that the danger of all currents depends on the good contact, skin resistance and the duration, since during the passage of the electric current tissue resistance drops markedly.

The course of the current through the body is of importance. Although there is a certain diffusion of electricity through the whole body, fatal effects are most often observed when the thoracic organs lie in the path of the current, or the thorax has been part of the contact. Where the contact occurs through the head, the symptoms may be merely burns, since nervous tissue does not seem to be easily affected. Where the leg and arm of the same side or both legs form the contact, the shock is rarely fatal. Hence in dislodging a victim the use of the foot is advisable.

Treatment.—Heart massage within five minutes of cessation has been said to be effective. With respiratory paralysis there is the strongest indication for artificial respiration, since, if this is kept up, heart action may recommence. Recovery of the fibrillating heart is very rare. Some authors advise intravenous adrenalin, strychnin or camphor, or even 5 per cent potassium chlorid. More recently intracardiac adrenalin with cardiac massage, or sometimes strong electric shocks to the precordium, have been advocated, in spite of their apparent danger.

At present it is generally felt that, although attempts at cardiac stimulation might be made, they must be brief and not interfere with artificial respiration, since every minute is of vital importance. Treatment must be immediately directed to the respiratory paralysis in the hope that spontaneous heart recovery may occur. A study of resuscitation methods has been made in behalf of the National Electric Light Association, in which strong recommendation is made in favor of the Schaeffer prone pressure method. First, however, the victim must be disengaged from the current either by switching out or by short-circuiting; if this is not possible, he must be removed. Would-be rescuers have often been involved in the fate of the first victim through carelessness. If no rubber gloves, or rubber shoes, or insulation is available, especially if the ground is wet, an attempt should be made to dislodge the victim or break the contact by some non-conducting object of wood or rubber. Failing this the hands should be wrapped in many thicknesses of dry cloth. When removed, artificial respiration must be begun immediately and, according to the above Commission, should be kept up for two hours if necessary, or until such secondary evidences of death as chilling of the tissues and lividity appear.

In view of the fact that correct performance is necessary, there are reproduced the instructions from the pamphlet prepared by the Commission on Resuscitation from Electric Shock:

1. As soon as the victim is clear of the live conductor, quickly feel with your finger in his mouth and throat and remove any foreign body (tobacco, false teeth, etc.). If the mouth is tight shut, pay no attention to the above-mentioned instructions until later but immediately begin resuscitation. The patient will breathe through his nose and, after resuscitation has been carried on a short time, the jaws will probably relax and any foreign substance in the mouth can then be removed. Do not stop to loosen the patient's clothing; *every moment of delay is serious.*

2. Lay the patient on his belly, one arm extended directly overhead, the other arm bent at elbow and with the face resting on hand or forearm so that the nose and mouth are free for breathing.

3. Kneel, straddling the patient's hips, with the knees just below the patient's hip bones or opening of pants pockets. Place the palm of the hands on the small of the back with fingers resting on the ribs, the little finger just touching the lowest rib, the thumb alongside of the fingers, the tips of the fingers just out of sight.

4. With arms held straight, swing forward slowly so that the weight of your body is gradually brought to bear upon the subject. This operation, which should take from two to three seconds, *must not be violent* as the internal organs may be injured. The lower part of the chest and also the abdomen are thus compressed, and air is forced out of the lungs, the diaphragm is kept in natural motion, other organs are massaged and the circulation of the blood accelerated.

5. Now *immediately* swing backward so as completely to remove the pressure. Through their elasticity, the chest walls expand, and, the pressure being removed, the diaphragm descends and the lungs are thus supplied with fresh air.

6. After two seconds swing forward again. Thus repeat deliberately twelve to fifteen times a minute the double movement of compression and release—a complete respiration in four or five seconds. If a watch or a clock is not visible, follow the natural rate of your own deep breathing; the proper rate may be determined by counting—swinging forward with each expiration and backward with each inspiration.

7. As soon as this artificial respiration has been started and while it is being continued, an assistant should loosen any tight clothing about the patient's neck, chest or waist. *Keep the patient warm.* Place am-

monia near the nose, determining safe distance by first trying how near it may be held to your own. Then the assistant should hit the patient's shoe heels about twenty times with a stick, and repeat this operation about every five minutes until breathing commences. Do not give any liquids whatever by mouth until the patient is fully conscious.

8. Continue artificial respiration without interruption (if necessary for four hours) until natural breathing is restored. Cases are on record of success after three and one-half hours of effort. The ordinary tests for death are not conclusive in cases of electric shock and doctors must be so advised by YOU, if necessary.

9. When the patient revives, he should be kept prone (lying down) and not allowed to get up or be raised under any consideration unless on the advice of a doctor. If the doctor has not arrived by the time the patient has revived, he should be given some stimulant, such as one teaspoonful of aromatic spirits of ammonia in a small glass of water, or a drink of hot ginger tea or coffee.

The patient should then have any other injuries attended to and be kept warm, being placed in the most comfortable position.

10. Resuscitation should be carried on at the nearest possible point to where the patient received his injuries. He should not be moved from this point until he is breathing normally of his own volition, and then moved only in a lying position. Should it be necessary, due to extreme weather conditions, etc., to move the patient before he is breathing normally, he should be kept in a prone position and placed upon a hard surface (door or shutter) or on the floor of a conveyance, resuscitation being carried on during the time that he is being moved.

11. A brief return of spontaneous respiration is not a certain indication for terminating the treatment. Not infrequently the patient, after a temporary recovery of respiration, stops breathing again. The patient must be watched and, if normal breathing stops, artificial respiration should be resumed at once.

This method is safe and effective, which cannot be said for many of the popular present-day mechanical devices for lung inflation. In addition it can be continued indefinitely, can be taught and applied immediately and gives a feeling of self-reliance unknown by those who depend upon patented mechanical devices. On account of its importance it should be taught to all police officials and those in authority. They should know the danger of delay and that many lives have been lost in transit to hospitals which could have been saved by immediate application of this simple procedure. Otherwise death occurs in from eight to ten minutes.

The after effects of shock on the nervous system are considered by most writers as psychogenetic. They are neuroses and no real pathology of the central nervous system has been discovered as a result of electrical action.

Prophylaxis.—Much can still be done in the prevention of accidents. The effective protection of workers by grounding and locking switches is a matter of engineering responsibility. Certain countries require specific safeguards for certain kinds of currents. In addition, education of the working forces is the strongest protection.

Electric Ophthalmia

The injury to the eyes called "electric ophthalmia" comes from exposure not only to welding but to short-circuit flashes. It is due entirely to the action of ultraviolet rays on the cornea and conjunctiva and has been discussed under the heading of Light.

Lightning Shock

Lightning is a spark discharge between celestial and terrestrial points of high potential difference. The effect on the eyes is that of other electric sparks, although it has been said to produce cataract, possibly from heat or molecular changes in the lens. Deafness is often caused by the concussion, an effect which is sometimes said to occur from concussion in short circuits. Lightning produces the clinical picture of any other form of electrical contact, although it often shows certain peculiarities of its own. One of these is a branching erythematous skin lesion, sometimes in the form of dilated capillaries, sometimes in actual edematous welts. The phenomenon is transient, the cause is unknown. There is a striking resemblance to the branches of trees. Another peculiarity of lightning is its violent concussive effect, shown on the clothing and the surrounding objects often without any burning or charring of the tissues. Often secondary injuries result from the disrupted fragments. The emotional shock is profound; psychoses, delirium and often persistent psychoneuroses follow recovery and recur especially at times of electric storm.

The hazard of the electromagnetic field for workers has also been a subject of discussion. Subjective sensations have been reported by many workers, but recent experimental work has shown that animals may be kept for a long time in the field of powerful electromagnets without any apparent effects on their well-being, and without production of pathology in their tissues.

In spite of the clinical observations, the knowledge of electrical action on tissues is still indefinite; and even in its serious and fatal consequences the effect of electricity on the organism still offers field for study.

FATIGUE

Fatigue has been defined by the British Health of Munitions Workers Committee as "the sum of the results of activity which show themselves in a diminished capacity for work." This definition, while not precise, may serve the practical purposes of a discussion of industrial fatigue. Activity of industrial workers is both physical and mental and so fatigue may be evidenced in a diminution of physical power or mental power or of both. Few actions involve physical activity alone and the interrelation of mental processes with physical renders the study of fatigue an intricate and difficult problem.

Though fatigue is a word popularly used somewhat indiscriminately and the production of fatigue in industry and elsewhere is often the occasion of condemnation of the circumstances producing the condition, it should be recognized that the development of fatigue is physiological until a state is attained when normal rest does not restore the fatigued individual to normal power. A fatigued man may well be a healthy one.

It has long been realized that fatigue is manifested both objectively and subjectively, though not of necessity in both manners simultaneously. With mental work subjective fatigue tends to develop before objective, while with physical exertion, especially if interest is maintained, objective fatigue may be evidenced before subjective.

If it be assumed that fatigue shows itself through lowered capacity for the doing of work, it is essential to the estimation of fatigue that the normal capacity be determined for each task considered. At the present time it is difficult to estimate fatigue either qualitatively or quantitatively. It is even more difficult, if not impossible, to differentiate fatigue due to industrial activity from that due to non-industrial factors. Within an industrial establishment the nature of the work done, the hours of labor, work methods and working conditions, such as posture, ventilation, illumination, all are related to fatigue production; while perhaps of equal importance are such non-industrial factors as food, housing, transportation, recreation, sleep, worries and personal habits. It must be true, however, that many of the latter "non-industrial" factors are definitely determined by industrial conditions and by wages received.

The Physiology of Fatigue

Muscular work involves the function of: (1) the muscles themselves; (2) the blood and the cardiovascular system; (3) the respiratory, alimentary and eliminative systems; and (4) the nervous and endocrine systems.

With muscular exertion catabolic processes increase and, as they exceed in activity the anabolic processes, products of catabolism accumulate. Lactic acid, for example, a product of muscular activity as well as an agent in that activity, is held, in part, in fatigued muscle tissue, but after long-continued muscular contractions is found in the blood and urine. Scott and Hastings have shown that there is an increased excretion of sulphur coincident with muscular effort, and Hastings has noted certain changes in the gaseous and osmotic relationships in the blood which suggest that with fatigue there is inability to maintain the "adjustments of the organism to the new conditions of energy release." Experimentally various substances, such as beta oxybutyric acid and skatol, may produce effects in muscle similar to those observed following prolonged use; but there is no conclusive evidence that any one substance is responsible for the production of fatigue. Despite the claims of Weichard, it has not yet been demonstrated that a "fatigue toxin" is the product of muscular exertion.

Though but little is known regarding the physicochemical aspects of fatigue, physiologists have made important contributions to the knowledge of sites of fatigue.

The stimulus for a muscular contraction originates either in the cerebral cortex or, more often, in a peripheral sensory end organ, passes either directly from the cerebrum to an anterior horn cell or, in the second instance (reflex arc), passes over an efferent fiber to the posterior horn, thence by an association fiber to a neuron of the anterior horn, through which an efferent impulse passes to the motor end organ in the muscle called upon to function.

There may, then, be considered the possibilities of fatigue of: (1) the sensory site; (2) the neuron body; (3) the nerve fiber; (4) the synapse or point of transmission of an impulse from nerve cell to nerve cell; (5) the motor end organ; (6) muscle tissue; (7) brain cells.

The neuron bodies have not been shown to be notably affected by use and for practical purposes they may be considered as not susceptible of fatigue.

A nerve trunk is known to be incapable of conducting an impulse within a very brief period following the passage of a previous impulse. This refractory period is of such a short duration (about .002 second) as to render it unlikely that it is concerned with the development of fatigue.

If in a frog nerve-muscle preparation the normal conduction of impulses from electric stimulation is blocked by the application of a galvanic current distal to the point of stimulation, applied over a period of hours, there may be obtained, after the withdrawal of the galvanic block, contractions of the muscles of such magnitude as to indicate that the long-sustained conduction of impulses from the original site of stimulation had not induced any important diminution in the power of conductivity of the nerve trunk.

In a spinal animal, one prepared for experimental purposes by decerebration, respiration being artificially maintained, a "scratch reflex" may be elicited by stroking the back or sides of the animal. If the stimulation is continued for some minutes, the reflex is no longer obtainable unless another site of stimulation is selected, in which event the response is as lively as that originally obtained. Sherrington presents evidence that the sensory site is not fatigable and it is evident that the motor end organs are fully functioning. The efferent and afferent nerves practically cannot be fatigued, hence it must be concluded that fatigue was developed at one or more of the synapses, the junctions between the several neurons of the reflex arcs involved.

At the terminations of motor nerves in muscle tissue are the neuromuscular junctional organs called motor end plates. A muscle may be stimulated, until contractions cease, to resume contraction when stimuli are applied directly to the muscle tissue. Fatigue is due in this instance to failure of the end organs.

Muscle tissue itself may evidence fatigue by progressive elevation of the threshold for stimulation. Pratt has shown that under stimulation the threshold for an individual muscle cell rises until the cell no longer responds, at which time it abruptly ceases to function. Up to this point it contracts with maximum power. A muscle in sustained action must be constituted of active and of refractory cells and possibly also of cells not yet called upon for service. It is apparent that in unusually heavy work more cells are used than in light work and if such activity be continued there must be interference with the normal protective and recuperative mechanism of the tissue, possibly through the introduction of some substance such as adrenin which may lower the threshold of irritability.

C. K. Drinker and K. R. Drinker, in discussing heightened resistance in synapses and myoneural junctions, state:

> "There is no doubt that in a willed motor act, such as repeated lifting of the hand and arm to a right angle with the body, fatigue eventually expresses itself, not because the muscles involved are exhausted, but because the impulses passing from the motor cortex to the muscles meet synapses which, as a result of repeated passage of impulses, have eventually become so refractory toward conduction that passage cannot be effected. Only two alternatives are then possible in order to reassume lifting. These are: (1) to increase the strength of the impulse so that it may break down the heightened synaptic resistance; or (2) to experience some sudden general influence which lowers synaptic resistance, thus permitting normal impulses to pass through and reach the muscles. It is safe to

say that the first of these never occurs. The nerve impulse, however started, has but one value. It is 'all or none.' No matter how great the willful effort, larger impulses in single nerve fibers cannot be manufactured. . . .

"While it is clearly far from the final truth, our present knowledge concerning the uncovering of unsuspected muscular power is entirely in terms of nerve endings and muscle, and does not concern the brain and spinal cord. There is good evidence that a sudden brace in the individual near exhaustion reflects direct changes in muscle cell and in the junctional area between the motor end plate and the muscle cell. . . . If we stimulate a nerve repeatedly and record contractions of the muscle which it governs, we shall eventually reach a condition in which the muscle will not respond to nerve stimulation, but will respond when stimulated directly. Many facts agree in localizing this failure in the myoneural junction, and it thus becomes apparent that between the brain, which may desire to drive a muscle to exhaustion, and the muscle itself, there is interposed a second chance for failure. There is first the possibility that impulses may fail through increased synaptic resistance in the paths within the central nervous system, and there is a second peripheral safeguard in the myoneural junction. There is good evidence that this second type of resistance can be broken down. Cannon and collaborators have shown that under certain circumstances there is secretion of adrenin into the blood-stream and that this substance is peculiarly adapted to bring about reactions favorable to the stressed individual. . . . It is not proper to think that adrenin betters the condition of the muscle. It simply renders the muscle incapable of failing to receive all the stimulation possible. Such a provision is manifestly favorable for a spurt but not necessarily favorable for the muscular future of the individual. It has not been shown experimentally whether muscles experiencing repeated return of irritability through adrenin injections may not eventually be driven to a state of fatigue, from which recovery is exceptionally difficult. . . . Frequently in individuals under great stress, we may see a somewhat protracted display of vigor which makes us feel that the human being in normal times operates far below his real capacity. It is clear, however, that such a conception may be quite untrue. An emergency results in reactions which permit the individual to use his muscles past the temperate safeguards which are normally imposed. It is, of course, highly advantageous to be able to transgress in this way in times of temporary need, but one must remember that the organism has been permitted to commit excess with the powers it possesses, not that it has been suddenly endowed with any real accession of power."

In summary, according to present knowledge, muscular fatigue involves affection of conductivity through synapses, of motor end plates and muscle cells. There is recorded no satisfactory evidence of changes in brain cells produced by long-continued mental activity.

Industrial Fatigue

Discussion of the physiology of fatigue must bring clearly to mind the importance of the restoration of a fatigued cell, member or individual to normal power. There is general popular recognition of "acute" fatigue, of weariness definitely related to exertion. There are less frequently noted the effects of long-continued labor, "cumulative" fatigue, function more persistently impaired because of inadequate restoration. In studies of industrial fatigue it is desirable to record evidences of fatigue in workers as manifested from hour to hour and from day to day, but it is of particular importance that there be sought evidences of diminution of capacity as it may be displayed over a period of months or years and as it may be expressed in terms of ill health.

Recognition of the importance of excessive fatigue in industry, not only because of its harmful effects upon the individual worker but as well because of its economic unsoundness, has led to the development of many measures for the estimation of fatigue.

The measures employed are frequently classed as direct or indirect tests and are related almost exclusively to the study of fatigue as manifested objectively. Subjective fatigue cannot be gaged with precision. Direct tests are purposed to measure changes in capacity in the individual through observations of selected body functions. Collis and Greenwood have emphasized the difficulties attending direct tests because of the almost unavoidable influence of subjective elements. A desirable direct test should be wholly objective.

Mosso's work with the ergograph laid the foundation for many subsequent studies of muscle power and the practical value of Maggiora's contributions regarding muscle activity and rest has only recently become evident in connection with the use of rest periods in industrial operation.

Two direct tests were used in the course of the fatigue studies made in American factories during the World War by the Committee for the Study of Industrial Fatigue operating through the United States Public Health Service.

The first was a procedure originally developed by Lovett and Martin for the study of muscle function in cases recovering from poliomyelitis, which has become known as "Martin's spring balance test." Traction, which is applied to overcome the resistance of various selected muscle groups, is measured by a self-recording spring balance. A method for the calculation of total strength is described. Martin states that daily variations in the strength records of individual workers tend to be reflected in their production records.

The second direct test employed by the Committee was Ryan's vascular skin reaction. Under proper conditions of illumination, using an instrument devised by Ryan and Gordon, two strokes are made on the skin of the anterior surface of the subject's forearm. With a stop watch is recorded the time elapsing from the completion of the first stroke until white streaks appear at the sites stroked. The "latent period" is succeeded by a "duration time" measured from the completion of the latent period until a blushing of the white streaks appear. According to Ryan and Gordon, the duration time is decreased with fatigue and lengthened by rest. King applied the test to soldiers with "irritable hearts" and found it of value in relation to the study of groups, though of less value in the consideration of individual cases.

Schneider has described a system of cardiovascular rating which was profitably employed as a measure of physical fatigue and efficiency during the War in the examination of aviators:

"1. The patient reclines for five minutes. (*a*) The heart rate is then counted for twenty seconds. When two consecutive twenty-second counts are the same, this is multiplied by 3 and recorded. The score is noted according to Part A in the following table. (*b*) The systolic blood-pressure is next taken by auscultation; two or three readings are made as a check.

"2. (*a*) The patient stands at ease for one or two minutes to allow the pulse to assume a uniform rate. When two consecutive twenty-second counts are the same, this is multiplied by 3 and recorded. The score is obtained by use of Part C in the following table. The difference between the standing and reclining pulse rates is scored then by use of Part B in the table. (*b*) The standing systolic pressure is next taken. The difference between this and the reclining systolic pressure is then scored by Part F in the table.

"3. The patient next steps on a chair, about eighteen inches high, five times in fifteen seconds timed by a watch. To make this test uniform he stands with one foot on the chair at the count one; this foot remains on the chair and is not brought to the floor again until after the count five. At each count he brings the other foot on the chair and at the count "down" replaces it on the floor. This should be timed accurately, so that at the fifteen-second mark both feet are on the floor. (*a*) Immediately, while he stands at ease, the pulse rate is counted for fifteen seconds; this is multiplied by 4 and recorded. (*b*) Counting is continued in fifteen-second intervals for two minutes, record being made of the counts at sixty, ninety and one hundred and twenty seconds."

The data from *a* will be scored by Part D in the table, taking the difference between this exercise pulse rate and the standing rate. The data in *b* are scored according to Part E in the table.

Schneider's System of Cardiovascular Rating

A—Reclining Pulse Rate		B—Pulse Rate Increase on Standing				
Rate	Points	0-10 Beats Points	11-18 Beats Points	19-26 Beats Points	27-34 Beats Points	35-42 Beats Points
50- 60	3	3	3	2	1	0
61- 70	3	3	2	1	0	—1
71- 80	2	3	2	0	—1	—2
81- 90	1	2	1	—1	—2	—3
91-100	0	1	0	—2	—3	—3
101-110	—1	0	—1	—3	—3	—3

C—Standing Pulse Rate		D—Pulse Rate Increase Immediately after Exercise				
Rate	Points	0-10 Beats Points	11-20 Beats Points	21-30 Beats Points	31-40 Beats Points	41-50 Beats Points
60- 70	3	3	3	2	1	0
71- 80	3	3	2	1	0	0
81- 90	2	3	2	1	0	—1
91-100	1	2	1	0	—1	—2
101-110	1	1	0	—1	—2	—3
111-120	0	1	—1	—2	—3	—3
121-130	0	0	—2	—3	—3	—3
131-140	—1	0	—3	—3	—3	—3

E—Return of Pulse Rate to Standing Normal after Exercise	
Seconds	Points
0- 60	3
61- 90	2
91-120	1
After 120:	
2-10 beats above normal...	0
11-30 beats above normal...	—1

F—Systolic Pressure, Standing Compared with Reclining	
Change in mm.	Points
Rise of 8 or more........	3
Rise of 2-7	2
No rise	1
Fall of 2-5	0
Fall of 6 or more	—1

"This scheme uses in part a plan proposed by Dr. J. H. McCurdy for rating infantry men in cardiovascular and neuromuscular efficiency. The scores for each of the six items range from + 3 to — 3. A perfect score, the sum of the value given to each of the six items, is 18."

[In analyzing data from the examinations of 54 aviators] "only 6 of the 54 cases had a score of 10 or better, while 88.8 per cent had scores ranging between 9 and — 1. These figures seem to indicate that a score of 9 or less is characteristic of physically unfit men."

Indirect tests of industrial fatigue are intended to gage fatigue as it may be indirectly manifested, as, for example, in output, in impaired health and in labor turnover.

Output.—Studies of the output of industrial workers offer what are probably the most satisfactory indirect tests of fatigue. They are, however, not wholly trustworthy, must be considered as suggestive rather than absolute indices and deductions from them should be guarded. Under industrial conditions as they are usually encountered, it is difficult to control experiments as intricate as those concerned with output. It is apparent from a glance at a chart prepared by Florence ("Schedule of Industrial Conditions with References to Determination of Working Capacity") that estimations of fatigue based upon output studies are not lightly to be undertaken.

CHART I

Schedule of Industrial Conditions with References to Determination of Working Capacity, Slightly Modified from Florence, after Drinker

I. Length and Intensity of Activity
II. Nature of Work: type of study available for estimation of fatigue
    A. Statistics output
    B. Accident incidence
    C. Power utilization
    D. Sickness and out-time, medical supervision
    E. Objective tests
III. Type of Workers
    A. Sex, age and race
    B. Experience; date of entering industry; former occupations
    C. Habits and home conditions
        1. The amount and use of earnings; thrift; food; diet and time of meals; alcohol; sleep and recreation; home accommodation and hygiene; support of dependents
        2. Method and length of transit from home to work
        3. Duties outside factory (housework of women, etc.)
        4. Sexual and family relations
    D. Point of view "Animus"; trade-unionism, patriotism, economic self-interest, herd instinct, etc.; general intelligence
IV. Factory Conditions
    A. Physical: time and place of work
        1. Air: temperature and humidity; ventilation and room space; dust and fumes; exhaust systems; smell
        2. Light; volume, concentration, glare
        3. Noise
        4. Safety devices and first aid

    5. Feeding
    6. Drinking water; rest rooms; bathing facilities
- B. Social and economic
    1. Flow of work; depressions and rush orders; routing
    2. Creation of staff. Appointment and dismissal. Permanency of tenure
       Unemployment. Instruction and supervision
    3. Maintenance of production
       Incentives; natural interest in work; scale, method and assurance of wage payment
       Discipline

It is desirable in comparing output rates under varying hours of work that there should be no changes in the personnel of the group observed; no changes in process, materials or speed of machinery; no changes in pay rates or radical variations in incentive to work; no changes in physical conditions, such as illumination and ventilation; and no observations made until practice effects have been obtained.

In Figure 6, copied from Collis and Greenwood, there are indicated hourly variations in output in diverse operations. The uppermost curve illustrates a close approximation to an ideal work curve.

Many studies have indicated that output for day workers usually reaches its maximum between ten o'clock in the morning and noon and is at its minimum near the end of the daily working period. It is probable that there is considerable hourly variation in the normal curve of capacity and there is need for extended observations upon many individuals before the normal capacity may be determined for any particular process.

Though output may show a decrease, it does not necessarily indicate physical deterioration of the individual. Many of the most involved studies of fatigue are lamentably lacking in consideration of changes in the physical condition of the operatives studied. Spaeth has pointed out the fact that it may be very difficult to detect the early stages of fatigue and that within certain limits production may increase with fatigue.

Daily variations have shown a stepping up of production from Monday until Thursday or Friday, occasionally with a drop on Friday. This may be explained in part as a practice effect, or the low production early in the week may be due to fatigue resultant from week-end activities.

Production curves for individual workers, kept over long periods, are of value as suggesting evidence of the effects of work on health or of health on production. Bunn studied the production curves of girls making incandescent lamps and was able to correlate low production with poor physical condition, production rising with an improvement of the health and with an increase in weight of the operatives concerned.

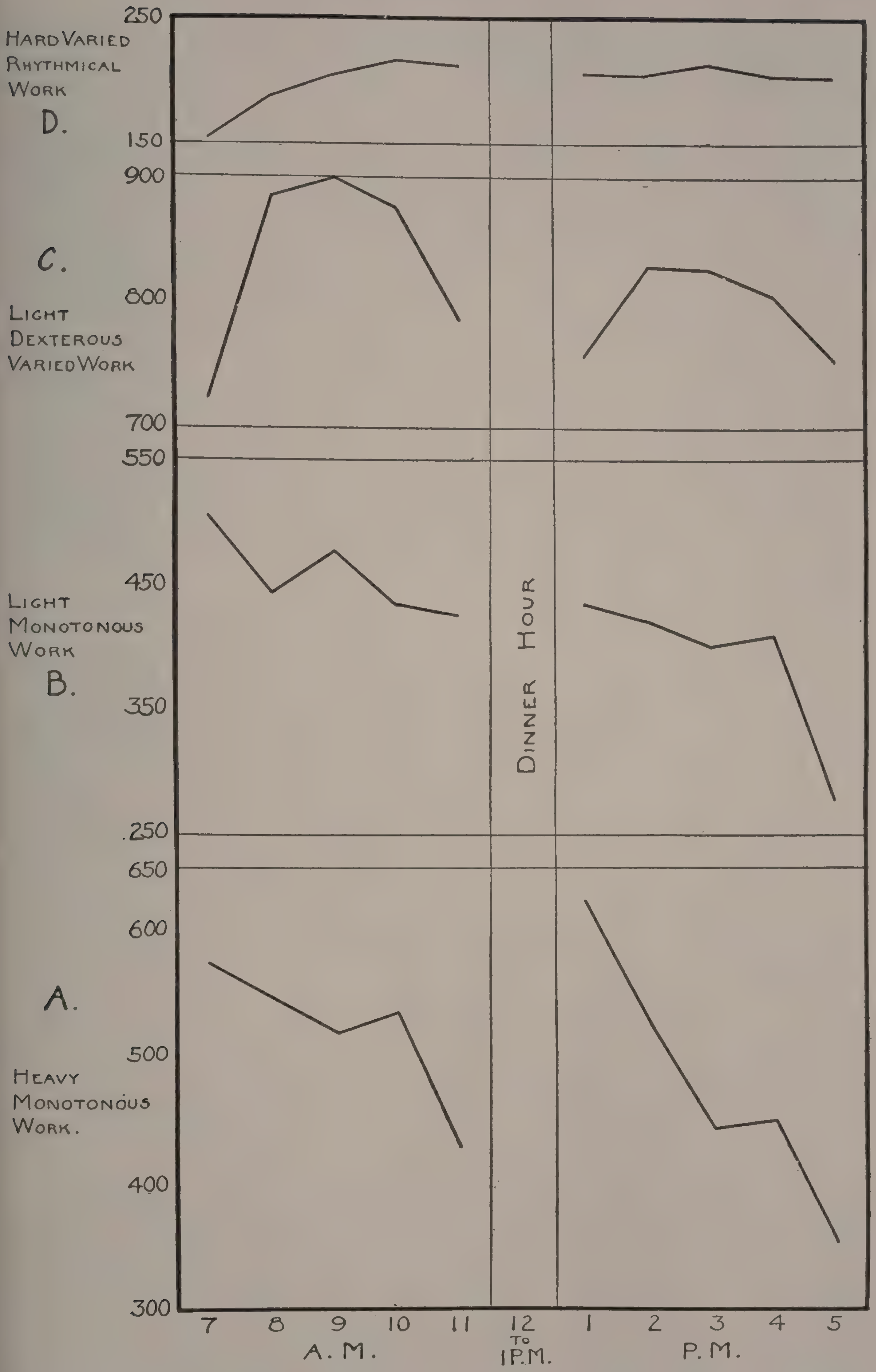


Fig. 6.—Hourly Output of Workers at Various Types of Processes. (After Collis and Greenwood.)

Lost time from sickness must be considered as an index of great value in studying the physical condition of workers under varying conditions of employment, as it is widely recognized that fatigue is one of the most potent factors in the causation of disease. It is unfortunate that at the present time few industrial organizations maintain records of sick absenteeism which warrant careful analysis. The United States Public Health Service and various associations of industrial physicians are now, however, endeavoring to standardize morbidity data and to collect statistical material which can scarcely fail to be of value not only in the study of working conditions but in connection with numerous problems of community health.

It is important that in considering sick absenteeism there be as little confusion as possible of absences due to real illness with those due to "personal" and other causes. For statistical purposes it is desirable, though frequently impracticable, that definite information be obtained regarding the causes of absences of even one day's duration, and for complete data it is essential that there be obtained, in addition, information regarding portions of days lost and tardiness, the "lost quarters" of the British studies.

Accuracy demands the careful investigation of individual cases and the recording of diagnoses made by competent persons. Unfortunately many circumstances may tend to invalidate the findings. For example, the sickness rate in an establishment which pays full wages during periods of sickness may vary greatly from that in a similar establishment paying no wages during illness. Different establishment sick benefit organizations have varying waiting periods during which no benefit is paid and this variation is not without its effect upon the duration of sick absenteeism. The day of the week upon which sick workers feel well enough to return to work seems to be dependent upon other conditions than disease and in general recovery is effected more often by Monday than by Friday or Saturday.

Wholly apart from such factors as these, but affecting industrial sickness rates, is the influence of epidemics, of communicable diseases in the homes of workers and climatic and other conditions bearing upon the incidence of incapacitating disease in the community at large. It is obvious that many other factors than fatigue are involved in the production of sick absenteeism.

There is a seasonal variation in sickness incidence which should be regarded in connection with all observations upon industrial morbidity rates. The form of the curve which represents such variations will vary with climatic conditions, but shows as well the influence of holidays and vacation periods. The curves on page 759 are representative.

Sickness incidence, like accident incidence, may bear an interesting relation to duration of employment, illustrated in the following chart based upon the sickness statistics of a large rubber manufacturing establishment. This relation is perhaps analogous to that existing between labor turnover, fatigue and duration of employment.

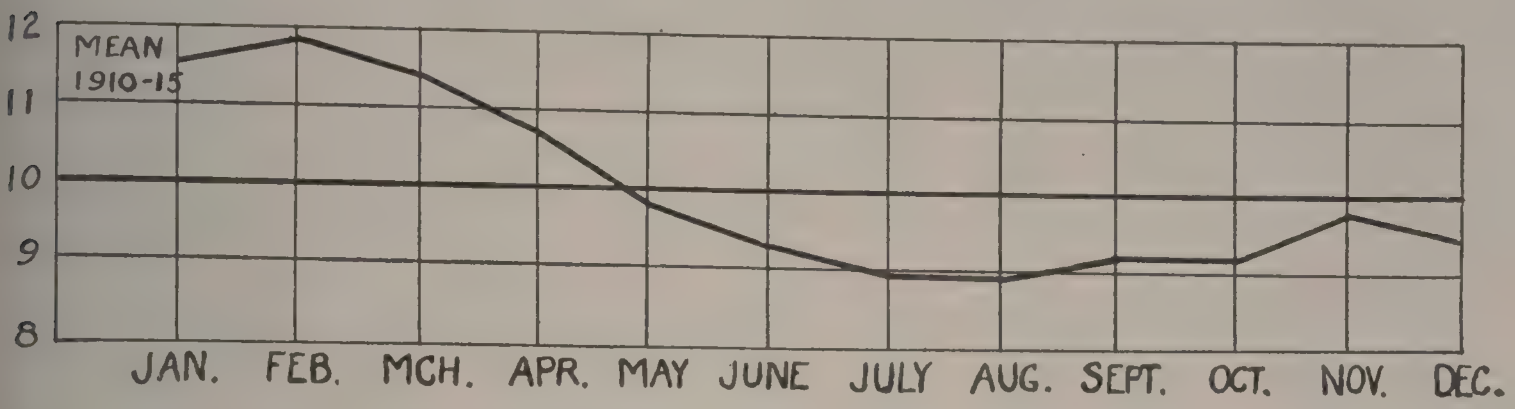


Fig. 7.—Seasonal Fluctuation of Sickness Rate. From statistics of large trade union sick benefit fund. Mean of means over six-year period. (United States Public Health Service.)

There has not as yet been demonstrated with any fair degree of precision the relation of excessive fatigue to lost time from sickness, though such a relation probably exists.

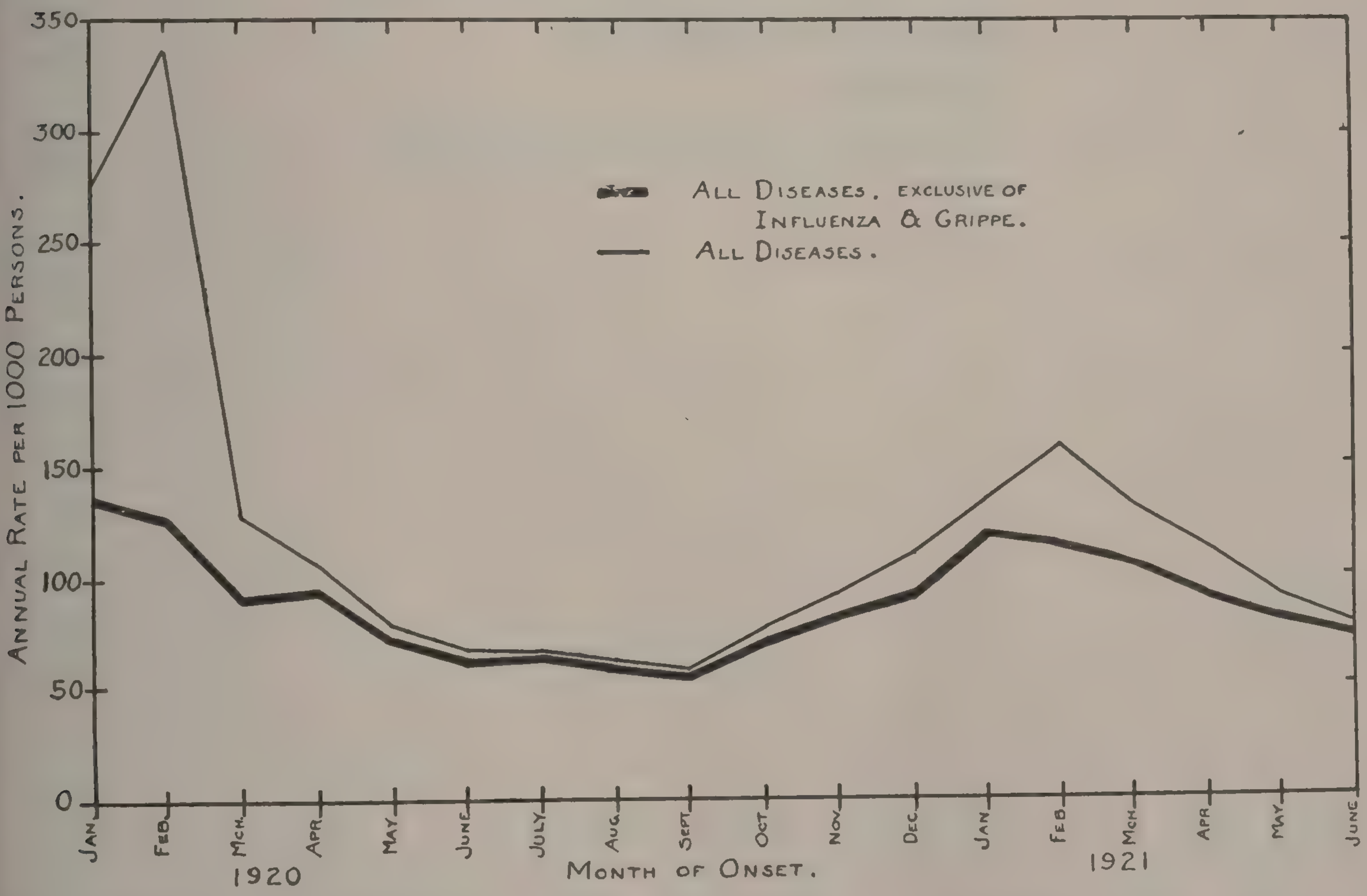


Fig. 8.—Seasonal Variation in Sickness Rate for All Diseases (Light Line) and All Diseases Exclusive of Influenza and Grippe (Heavy Line). From statistics of sick benefit associations. Includes only cases of sickness and non-industrial accidents which caused absence from work for one week or longer. (United States Public Health Service.)

A great number of contributions to the literature have been concerned with the inter-relation of fatigue and industrial accidents. It was Vernon's opinion that "speed of production is the essential factor in accident causation which can never be neglected." An increase in the rate of production may bring about a relatively greater increase in the number of attendant accidents. Collis and Greenwood observe that "increase of accidents is then primarily an indication of an increase rather than a decrease in working capacity of activity rather than fatigue." Vernon held, however, that under certain conditions fatigue was productive of accidents. He found that with women working night shifts, accidents were 47 per cent more numerous on the last day of the week than on the first, that accidents to women were two and a half times more numerous during a 12-hour day, 75-hour week, than during a 10-hour day, 54½-hour week.

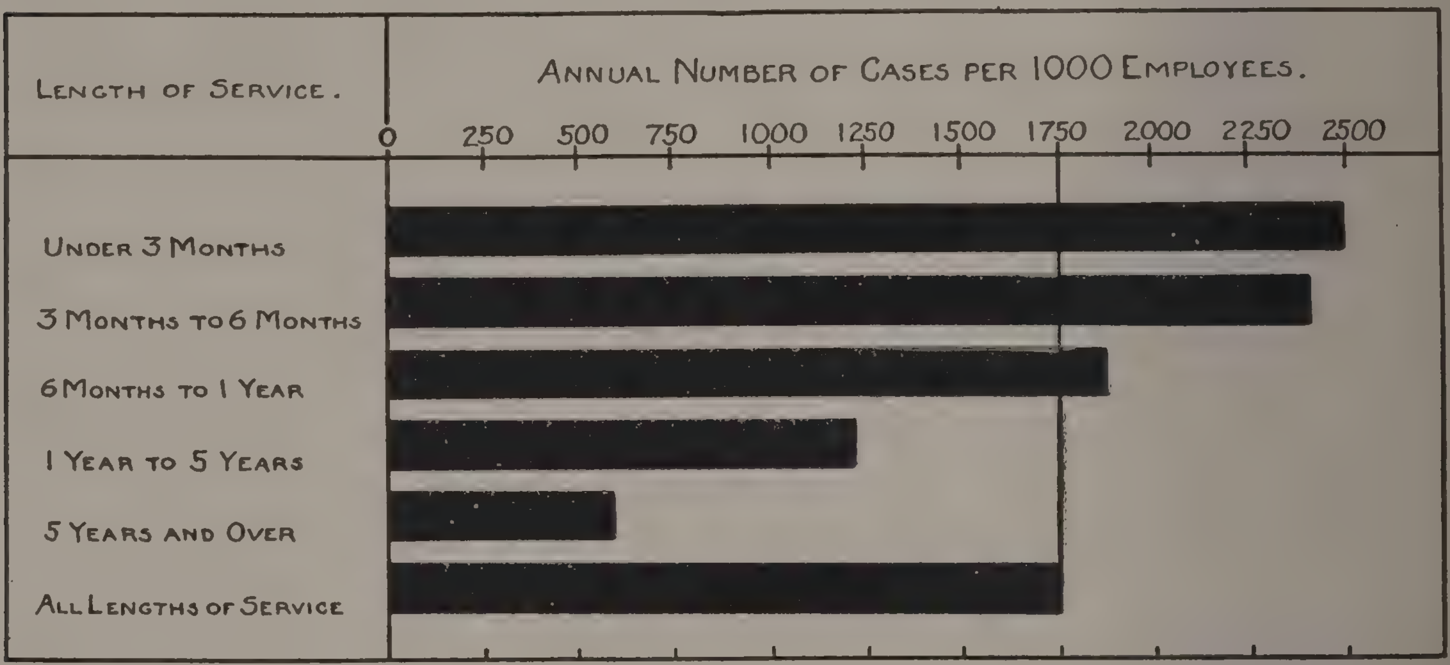


Fig. 9.—Frequency of Sickness and Non-industrial Accidents over Two-year Period Classified According to Length of Service with the Employing Company. From the statistics of a large manufacturing establishment, including cases causing absence from work for from one day to one year. (United States Public Health Service.)

The accident rates for men show no important change under similar variations in working hours.

The conclusions regarding industrial accidents of the American Committee on Industrial Fatigue of the Council of National Defense follow:

"*a.* In the absence of fatigue, accidents vary directly with speed of production, owing to increased exposure to risk.

"*b.* The breaking up of this regular variation by fatigue is indicated by:

"(1) The rise of accidents with the fall of output.

"(2) The disproportionate rise of accidents with the rise of output and the absence of a proportionate fall of accidents with the fall of output in the final hours of the day.

"*c.* The importance of fatigue in the causation of accidents is emphasized by the fact that the higher accident risk accompanies the deeper decline of working capacity:

"(1) In the second spell as compared with the first.

"(2) In muscular work as compared with dexterous and machine work.

"(3) At the 10-hour plant as compared with the 8-hour plant.

"*d.* The level of the accident rate varies inversely with the experience of the workers."

Spoilt work offers indirect evidence of fatigue, though accurate data bearing upon this subject are not abundant. Damage to products or tools probably occurs at about such times as accidents to workers are incurred and is undoubtedly due in part to the speed of production and not wholly to failing muscular coördination evidencing fatigue. It has been found that the error curve, among clerical workers, varies inversely with the output, that efficiency may be measured by output and that the form of the error curve is similar to that due to physical fatigue.

Labor turnover is unquestionably related to fatigue, though it is difficult to recognize in a coefficient of turnover a definite index of fatigue. New employees frequently find themselves unable to maintain a prescribed pace, doing work to which they may not be accustomed. The work may be such as to demand at first excessive effort of mind and body and workers leave the new-found jobs for others which promise greater ease. It is recognized that labor turnover is greatest among employees of short periods of service.

There are certain factors which are of special importance in the production of industrial fatigue and which are, in consequence, worthy of consideration because of their bearing upon the prevention or reduction of excessive fatigue.

Hours of Labor.—Some decades ago industrialists made frequent declaration that it was in the last hour of work that profits were made, that "the longer the hours, the greater the profit." In different phrasing the same concepts are expressed even now, but they are generalizations applicable only within certain industries and to certain types of operation.

An extensive experience with munition workers during the War led Vernon to state, "that the hours of labor should be varied between wide limits according to the character of the work performed. This obvious fact is not realized by many managers of munition works, and the tendency is usually toward uniformity of hours for all types of labor and for workers of both sexes." The truth of this statement should be held in mind in considering the proper length of the workday in any industry. There are few industrial establishments in which the processes of manufacture are so uniform in character as to justify the establishment of workdays of the same length for all workers, upon the assumption that such hours would provide for maximum output with minimum fatigue. The question of a 48-hour week, for example, is not alone one of fatigue but is a complex social, economic and human problem. Scientific analyses of statistics of output and sickness under varying hours of work are of value, but they can seldom serve as a foundation for arguments favoring a short workday.

Output studies have shown that in many processes in which production is not almost wholly determined by machinery, as regards both quantity and quality, a reduction of working hours is accompanied by an increase in production possibly so great as to more than compensate for the decrease in hours worked.

The following table based upon material published by Vernon presents in condensed form the results of the reduction of the hours of work, expressed in terms of output.

Results of Reduction of Hours

Nature of Work	Week Ending	Actual Hours of Work per Week	Relative Output per Working Hour	Hours of Work by Relative Output	Per Cent Reduction of Hours	Per Cent Gain or Loss in Output
Men sizing fuse bodies. Heavy work.	Nov. 14 to Dec. 19	58.2	100	5820		
	Feb. 27 to Apr. 16	50.5	122	6161	13	+ 6
	Oct. 7 to Nov. 4	51.3	137	7028	12	+21
	Nov. 11 to Dec. 16	51.2	139	7117	12	+22
Women turning aluminum fuse bodies. Moderately heavy work.	Nov. 14 to Dec. 19	66.2	100	6620		
	May 14 to July 2	54.8	134	7343	17	+11
	Nov. 11 to Dec. 16	45.6	158	7205	31	+ 9
Women milling a screw thread. Light work.	Nov. 21 to Dec. 19	64.9	100	6490		
	July 16 to Aug. 6	54.8	121	6631	16	+ 2
	Oct. 14 to Nov. 18	48.1	133	6397	26	— 1
Boys boring top caps (day and night shifts). Light work.	Nov. 15 to Dec. 19	72.5	100	7250		
	May 1 to May 28	54.7	117	6400	25	—12
	July 3 to Sept. 23	52.8 } 54.5	129 } 129	7030	25	— 3
	July 2 to Sept. 23	56.2	128			
Boys sizing base plugs. Moderately heavy work.	Nov. 22 to Dec. 19	68.3	100	6830		
	Feb. 7 to Apr. 16	57.0	142	8094	17	+19
	May 7 to May 31	60.9	155	9440	11	+38

The table offers striking evidence of the falsity of the traditional belief in the economic value of long hours of labor, certainly so far as these particular processes or similar ones are concerned. The first group of men, working seven hours less a week, increased their weekly output 22 per cent. The second group of women, working over twenty hours less a week, increased their weekly output 9 per cent. The third and fourth groups, employed on light work, with a reduction of hours amounting to about 25 per cent, almost maintained the production of the long work period, the hourly output showing a marked increase. The fifth group displayed a remarkably favorable reaction to the shortening of hours of work, a reduction in hours of 11 per cent, resulting in an increase in output amounting to 38 per cent.

Vernon made use of a graphic method for the estimation of probable rates of output, assuming reduction in weekly hours beyond those for which data were actually available. Though somewhat speculative, estimates of this character are of interest and value. Figure 10 represents graphically data set forth in the foregoing table, the broken lines showing the probable trend of the respective output curves.

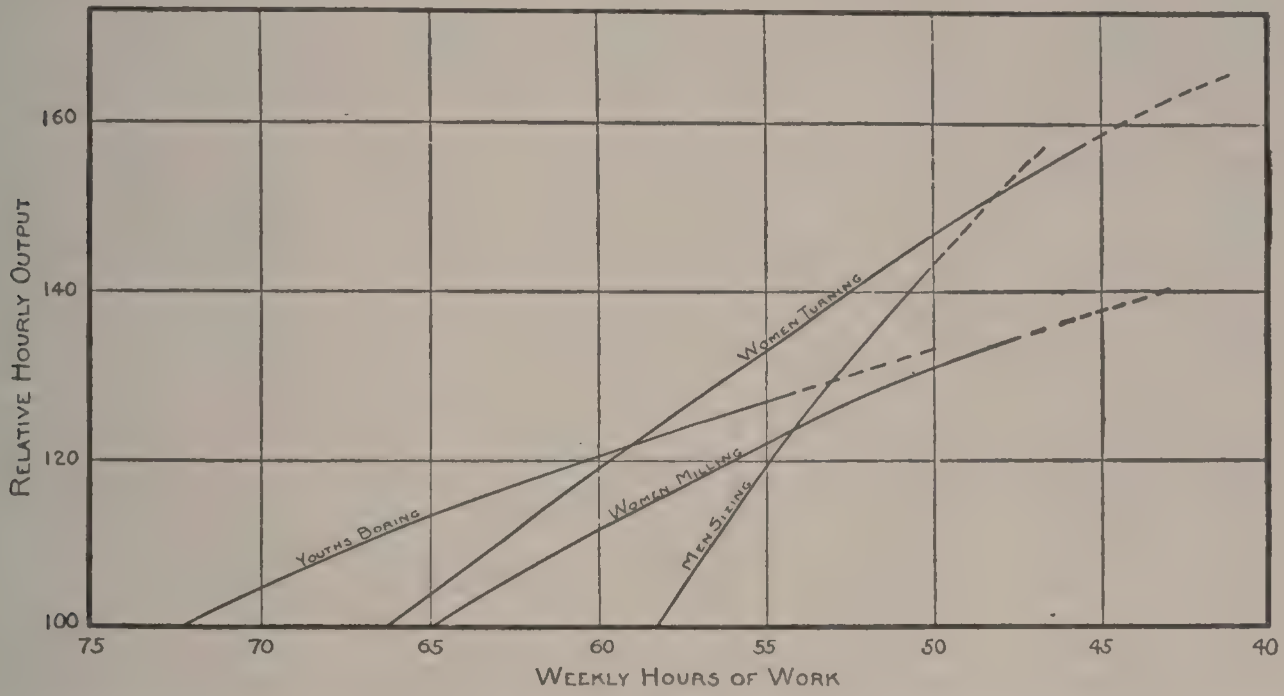


Fig. 10.—Results of Reduction of Hours. The broken lines show the probable trend of the respective output curves. (After Vernon.)

Rest Periods.—There is much evidence that in certain industries the introduction of rest periods favors an increase in production without an accompanying fatigue of workers. The rests may be frequent and of short duration or longer, periodic recesses. Frederick Taylor's illustration of the value of frequent rest periods in the handling of pig iron is of much interest. An average workman had loaded 12½ long tons of pig iron daily. At this task, a workman, acting under continued instructions as to when to work and when to rest, was able to load 47½ tons, and maintained this capacity over a period of years. Other workmen similarly instructed displayed similar abilities.

The report of the American Committee on Industrial Fatigue indicates that, in the plants studied by that committee, the hourly rate of production was usually increased after recesses, while the total daily production varied, with some workers falling and with others being maintained. There was noted an increase in production following holidays. It was the opinion of Dr. Vernon that rest periods are essential in active work and, if not provided for, are taken spontaneously by workmen.

Night Work.—The British Committee concluded that night work for men, if not continuous, did not result in any decrease of production. With women night production fell very slightly below that of the day. With continuous night employment, however, the night production of women was notably inferior to that of the day. The Committee attributed the inferiority of continuous night work to the probable difficulties workers experienced in securing proper rest during the day and to the devotion of resting hours to recreation and home duties.

Though it has been customary to employ night shifts for longer hours than day shifts, it is in many industries a measure of doubtful economic value, wholly aside from its social aspects. The American Committee found in a plant maintaining a 10-hour day shift and a 12-hour night shift that there was an abrupt fall of production in the last two hours of the night shift and a gradual slowing in rate of production during the night.

Temperature, Humidity and Ventilation.—The relation of temperature to the physical resources of workers, as evidenced in output, is apparently established, though as yet statistical evidences are meager. The exhausting effects of high temperatures, especially when combined with high humidity and limited air movement, are generally recognized.

Seasonal variation in output shows a striking agreement with the seasonal variation in temperature. The accompanying figure (Fig. 11), copied from a report by Dr. Vernon, upon the effects of hours of work and ventilation upon output in the tin-plate industry, illustrates this point clearly. It should be noted, in this connection, that the period of low production coincides in general with the period of low morbidity.

Among British munition workers, accident rates, as indicative of the effects of temperature upon operatives, tended to increase as the temperature fell below the optimum range of 65° to 69° F. and again increased as the temperature rose above 70° F.

Lighting is related to industrial fatigue in so far as inadequate or improper illumination serves to render work more difficult and to demand unaccustomed care and application. Though "eye-strain" is a term exceedingly difficult of definition, it is freely used in the discussion of problems of industrial illumination. There is reason to believe that, except under extreme conditions of illumination and work, eye-strain is not produced unless there is preëxistent some disturbance of ocular function. It has been urged that the efficiency of employees can be materially advanced by the painting white of the walls of their workrooms; but it would be of more fundamental value to secure first proper correction of refractive errors in those possessed of ocular defects.

"Scientific management" has progressed so far as to demonstrate the faults of many accepted, traditional work methods. Through time and motion studies the waste of energy in a multitude of tasks has been exposed and better methods suggested. Though the "one best way to work" may not have been found by Taylor and his followers, it is certain that they have evolved better ways to work and their contributions toward the diminution of unnecessary and excessive industrial fatigue have been invaluable.

Not only are the motions which a worker makes a source of fatigue, but his posture at work is of great importance in this connection. The proper adjustment of the working planes in relation to the individual worker and the process at which he is engaged and, if at a sitting job, his chair or stool, warrant thoughtful consideration.

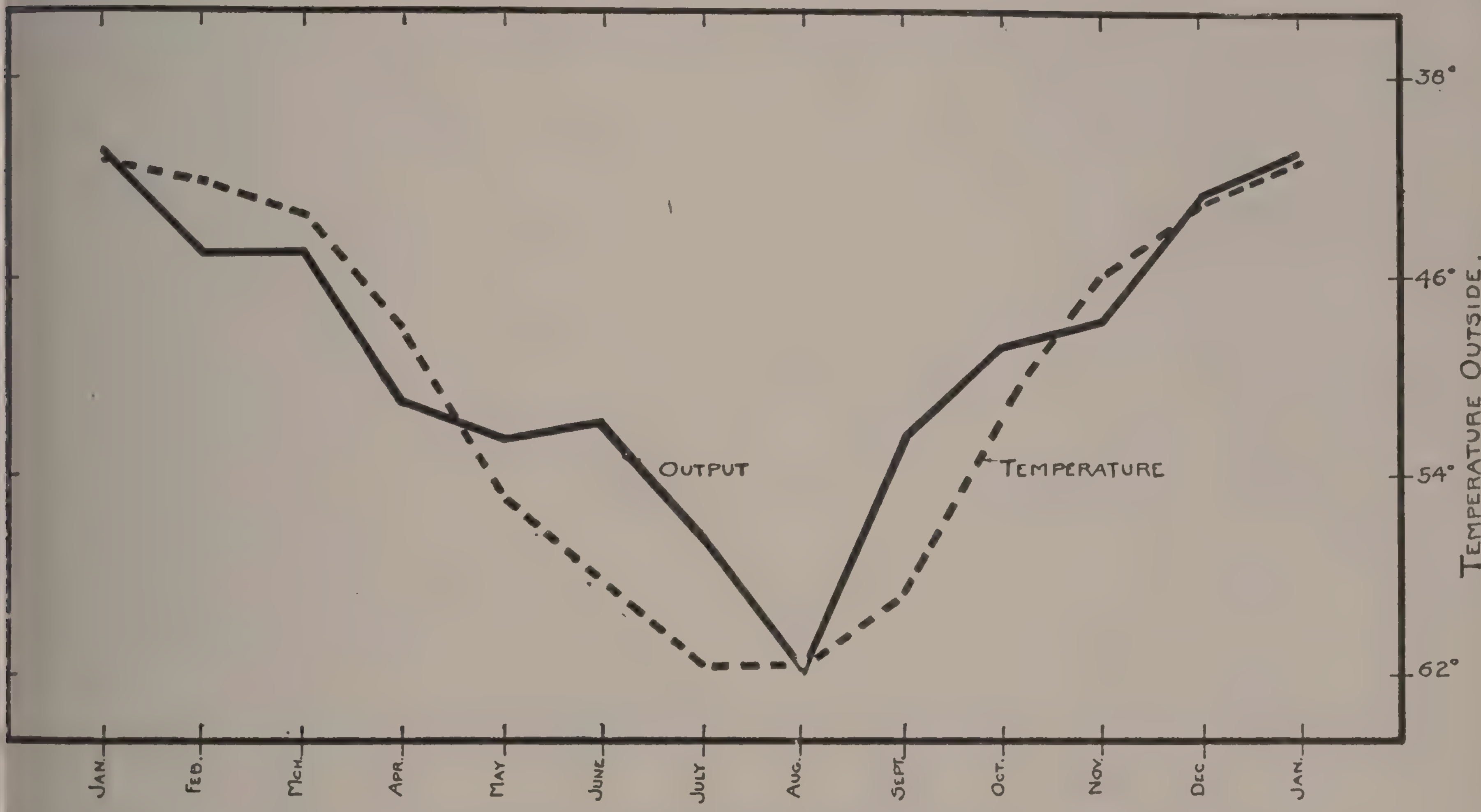


Fig. 11.—Seasonal Relations of Temperature and Output. (After Vernon.)

During recent years, a popular interest in industrial seating has evoked considerable hastily prepared literature upon the subject and has resulted in the flooding of markets with "orthopedic" chairs of great variety. It must be recognized that even chairs of good design do not offer a panacea for the postural ills of industrial workers. The harmful effects of constant standing are matched by the harmful effects of constant sitting. Through the designing of work benches to permit of standing or sitting work, through rest periods, through arrangements whereby workers carry finished work away or bring supplies, posture may be varied.

The maintenance of proper posture during work may be facilitated by the proper design of work bench or machine, by a good seat when the use of a seat is practicable, by a suitable foot rest or treadle and by convenient location of supplies and the products of the operation.

Of necessity all of these factors must be considered in their relation to individual workers. For a factory in which are carried on a variety of operations there is no "ideal" work chair or bench. Benches may be adjustable or supplemented by floor platforms for workers of short stature or bench platforms for unusually tall workers. Work chairs to meet the demands of the customarily greatly varied types of workers who are to use them must either be supplied in a variety of sizes and forms or be adjustable. Chairs of the latter sort are, for economic reasons, more desirable. The height of the seat above the floor, the depth, shape and mobility of the seat, the form and position of the back are of importance in considering chair design. Stability, finish, sturdiness and cost are in addition of concern to industrial executives. A chair may be possessed of many merits and carry the endorsement of eminent medical authorities and yet be wholly unsuited to the requirements of a particular operation. A problem in industrial seating should be carefully worked out with regard for such requirements and a chair found or made to suit the machine and its operator. In relatively few instances are machines designed with much thought of the proper position of the operator upon whose functioning the utility of the machine must depend.[8]

Occupational Neuroses.—Under the somewhat unsatisfactory term "neuroses" have been grouped a variety of conditions associated with the sustained use of limited groups of muscles. Of this nature are the "cramps" of telegraphers, penmen and pianists and the disabilities incurred by cigar makers and hammermen. Many of the cases of occupational neuroses present a large psychic element; in others there are associated with the local symptoms and signs evidences of preëxistent disease, such as the cervical hypertrophic arthritis frequently found in cigar makers. Though the relation of the various disabilities to monotonous and continued movements may be beyond question, there is little evidence that the neuroses are truly fatigue effects.

The elimination or reduction of industrial fatigue is essentially a problem of the mutual adaptation of a workman and a job with regard for the reasonable demands of the job and physical and mental resources of the worker. Many studies have revealed the enormous incidence of physical defects and impaired function among men of working age in this and other countries. Without consideration of such defects and of changes in the physical condition of workers under observation, fatigue studies cannot but be lamentably lacking.

---

[8] The New York Industrial Commission Bulletin 104, 1921, "Industrial Posture and Fatigue," presents an interesting and popular discussion of the subject and contains a bibliography.

The treatment for fatigue is rest. Various devices for securing rest have been considered in the foregoing discussion of the conditions under which industrial work is carried on.

There is probably no factor within and without industrial life as important in its causative or precipitative relation to disease as fatigue. It is "the great industrial disease." Though it is difficult of accurate diagnosis, experience has brought forth some knowledge of its causes, of its prophylaxis and its treatment. It still remains the most important field of endeavor for the industrial hygienist.

REFERENCES

COMPRESSED AIR ILLNESS, OR CAISSON-DISEASE

Bassoe, P. Tr. XVth Internat. Cong. Hyg. & Demog., iv, 626, Washington, 1913.

Bert, Paul. La Pressure Baromtrique, Paris, 1878.

Boycott, A. E., and Damant, G. C. C. Journ. Hyg., viii, 342, 445, 1908.

Burwell, G. A. U. S. Bureau Mines, Technical Paper No. 11, Washington, 1912.

Erdman, Seward. Kober and Hanson, Diseases of Occupation, Philadelphia, 1916.

Haldane, J. S. Report of Board of Trade on Ventilation of Metropolitan Railway Tunnels, Parliamentary Papers 8684, London, 1897.

——— Report to the Lords Commissioners of the Admiralty on Deep Water Diving, London, 1907.

——— Journ. Soc. Arts, xvi, 214, 1908.

Haldane, J. S., and Priestly, J. G. Journ. Physiol., xxxii, 225, 1905.

Heller, Mager, and von Schrötter. Luftdruck Erkrankungen, Vienna, 1900.

Henderson, Y., and others. Journ. Indust. Hyg., iii, 79, 137, 1921.

Hill, Leonard. Caisson Sickness, Arnold, London, 1912.

Hoppe-Seyler, F. Müller's Arch., 63-75, 1857.

Japp, Henry. Tr. XVth Internat. Cong. Hyg. & Demog., iv, 639, Washington, 1913.

Keays, F. L. Compressed Air Illness, New York, 1909.

Levy, Edward. U. S. Bureau Mines, Technical Paper No. 285, Washington, 1922.

McWhorter, J. E. Am. Journ. Med. Sc., cxxxix, 373, 1910.

Pol and Watelle. Ann. d'hyg., after L. Hill, 2d series, i, 241, 1854.

Snell. The Compressed Air Illness or Caisson Disease, London, 1896.

Triger. Compt. rend. de sciences, after L. Hill, xiii, 884, 1841.

Vernon, H. M. Proc. Royal Soc., lxxix, B, 366, London, 1907.

Heat, Humidity and Cold

Bramwell, B. Edinb. Med. Journ., xii, 387, 1914.

British Health of Munitions Workers Committee, Memorandum No. 9, London, 1916.

Cicconardi. Arch. per le sc. med., xliii, Nos. 5, 6.

——— Abstr. in Lavoro, xii, 106, 1921.

Edsall, D. L. Am. Journ. Med. Sc., cxxviii, 1003, 1904.

——— Journ. Am. Med. Ass., li, 1969, 1908.

Effect of the Air Hammer on the Hands of Stone Cutters, Bull. U. S. Bureau Lab. Statistics, No. 236, Washington, 1918.

Fiske, C. N. Tr. XVth Internat. Cong. Hyg. & Demog., iv, 655, Washington, 1913.

Gaisbock, F. Wien. Arch. f. inn., Med., No. 1, 2, 1921.

——— Abstr. in Journ. Am. Med. Ass., lxxviii, 1094, 1922.

Harrington, D., and McElroy, G. E. U. S. Bureau Mines, Rep. of Investigations, Serial No. 2355, Washington.

Hill, Leonard. The Science of Ventilation and Open Air Treatment, Part I, Med. Research Comm., Special Rep., Series No. 32, London, 1919; Part II, Special Rep., Series No. 52, London, 1920.

Hill, L., and Greenwood. Internat. Journ. Pub. Health, ii, 232, 1921.

Hill, Leonard, Griffith, O. W., and Flack, M. Tr. Roy. Soc. London, Series B, ccvii, 183.

Orenstein, A. J., and Ireland, H. I. Journ. South African Inst., England, 1921. Cited by Harrison and McElroy.

——— Journ. Indust. Hyg., iv, 30, 70, 1922.

Sayers, R. R., and Harrington, D. U. S. Pub. Health Service, Pub. Health Rep., xxxvi, 116, 1921.

Watkins, J. A. U. S. Pub. Health Service, Pub. Health Rep., 2111, 1917.

White, R. Prosser. Occupational Affections of the Skin, 2d ed., New York, 1920.

Widal, F., Abrami, P., and Brissaud, E. Compt. rend., clxxiii, 207, 1921.

Light

Bell. Journ. Indust. Hyg., ii, 133, 1920.

Clark. Physiological Action of Light, Physiol. Rev., ii, 277, bibliography, 1922.

Crookes, Phil. Trans., Series A, ccxiv, 1, 1914.

Dorno. Physik der Sonnen und Himmelstrahlung, Braunschweig, 1919.

Eastman. Tr. Illuminating Engineering Soc., xv, 1, 77, 1920.

Eye Conservation in Industry in "Waste in Industry," Federated American Engineering Societies, 1922.

First Report of Miners' Nystagmus Committee, bibliography, Med. Research Council, Publication No. 65, London, 1922.
Gould. XVth Internat. Cong. Hyg. & Demog., Sec. iv, 582, Washington, 1913.
Hecht. Journ. Physiol., iii, 1, 285, 367, 375, 1920.
Hess, A. F., and Lundagen. Proc. Soc. Exper. Biol. & Med., xix, 380, 1922.
Hess, C., and Parsons, J. B. XVIIth Internat. Cong. Med., section in Ophthal., bibliography, 1913.
Hibben. Factory, xxviii, 5, 523, 1922.
Hoffmann. Bulletin 93, U. S. Bureau Mines, 1916.
Legge, T. M., and others. Report on Cataract in Glass Workers, British Home Office, 1907.
Report of the Glassworkers' Cataract Committee, British Home Office, London, 1920.
Robinson. Brit. Med. Journ., i, 191, 1903.
——— IId Internat. Cong. Indust. Dis., Brussels, 1910.
Coblentz and Emerson. U. S. Bureau of Standards Technical Paper No. 93, 1919.
Verhoeff and Bell. Science, xl, 452, 1914.
——— Proc. Am. Acad. Arts & Sc., li, 630, 1916.
Vogt. Arch. f. Ophth., lxxxi, 155, 1912; lxxxiii, 99, 1914.

Radiation

British Med. Journ., i, 936, June 25, 1921.
Colwell and Russ. Radium, X-rays and Living Cell, Bell, London, 1915.
Degrais. Paris méd., xii, 291, April 8, 1922.
Gudzent and Halberstaeder. Deutsche med. Wchnschr., xiii, 633, March 26, 1914.
Jaeger. München med. Wchnschr., lxix, 821, 1922.
Kaye. X-rays, Longmans, London, 1918.
Ledoux-Lebard. Paris méd., xii, 299, April 8, 1922.
Loeb, L. B. Journ. Radiol., ii, 1, July, 1921.
——— Am. Journ. Roentgenol., ix, 497, 1922.
Ordway. Journ. Am. Med. Ass., lxvi, 1, January, 1916.
Pfahler. Am. Journ. Roentgenol., ix, 647, 1922.
Porter, C. A. Journ. Med. Research, xvi, 357, 1909.
Rutherford, E. Radioactivity, Cambridge, 1905.
Soddy. Interpretation of Radium and Structure of Atoms, G. P. Putnam's Sons, New York, 1920.
Solomon. Paris méd., 96, February 4, 1922.
Wolbach. Journ. Med. Research, xvi, 415, 1909.

Electricity

Brit. Med. Journ., i, 478, 1922.
Drinker, C. K., and Thompson, R. M. Journ. Indust. Hyg., iii, 117, 1921.
Jellinek, S. Elektropathologie, Ferdinand Enke, Stuttgart, 1903.
——— Atlas der Elektropathologie, Urban & Schwarzenberg, Wien, 1909.
——— Wien. klin. Wchnschr., xxxiv, 20, 239, 1921.
Jex-Blake. Brit. Med. Journ., i, 425, 548, 601, 1913.
Oliver, Sir Thomas. XVth Internat. Cong. Hyg. & Demog., Sec. iv, 703, Washington, 1913.
Proc. Roy. Soc. Med., xv, 45, 1922.
Report of Commission on Resuscitation from Electrical Shock, National Electric Light Association, 1913, Third Commission, 1918.
Weiss. XVth Internat. Cong. Hyg. & Demog., Sec. iv, 730, Washington, 1913.

Fatigue

Baumberger, J. P. Journ. Indust. Hyg., iii, 149, 1921.
British Health of Munitions Workers Committee, Memorandum No. 12, London, 1916.
——— Report by T. Loveday, Causes and Conditions of Lost Time, London, 1917.
——— Memorandum No. 21, Final Report, London, 1918.
Bunn, W. H. Journ. Indust. Hyg., iv, 93, 1922.
Cannon, W. B. Bodily Changes in Pain, Hunger, Fear and Rage, D. Appleton & Co., New York, 1915.
Collis, E. G. and Greenwood, M. The Health of the Industrial Worker, Blakiston, Philadelphia, 1921.
Drinker, C. K., and Drinker, K. R. Physiology and Pathology of Work, Oxford System Med., iv, 503, 1921.
Fletcher, W. M., and Hopkins, F. G. Proc. Roy. Soc. London, Series B, lxxxix, 444, 1915-1917.
Forbes, A. Physiol. Rev., ii, 361, 1922.
Hastings, A. B. U. S. Pub. Health Service, Pub. Health Bull. No. 117, Washington, 1921.
——— U. S. Pub. Health Service, Pub. Health Rep., xxxiv, 1682, 1919.
King, J. T., Jr. Arch. Int. Med., xxiii, 527, 1919.
Lee, F. S. Harvey Lectures, 1905-1906, Lippincott, Philadelphia, 1906.
Lovett, R. W., and Martin, E. G. Am. Journ. Orthop. Surg., xiv, 415, 1916.
Lucas, K. The Conduction of the Nervous Impulse, Longmans, London, 1917.

Martin, E. G. Physiol. Rev., i, 454, 1921.

——— Am. Journ. Physiol., xlv, 538, 1918; xlvi, 67, 1918.

Martin, E. G., and Rich, W. H. Ibid., xlvii, 29, 1918.

Meeker, Royal. U. S. Bureau Lab. Statistics, Month. Lab. Rev., No. 3, ix, 1, Washington, 1919.

Mosso, A. Fatigue, translated by M. and W. B. Drummond, New York, 1906.

Pratt, F. H. Am. Journ. Physiol., xliii, 159, 1917; xliv, 517, 1917.

Ryan, A. H., and Gordon, J. H. Ibid., xlv, 537, 1918.

Ryffel, J. H. Journ. Physiol. Proc., No. 29, xxxix, 1909-1910.

Schneider, E. C. Journ. Am. Med. Ass., lxxiv, 1507, 1920.

Scott, E. L., and Hastings, A. B. U. S. Pub. Health Service, Pub. Health Rep., xxxv, 2445, 1920.

Sherrington, C. S. The Integrative Action of the Nervous System, Charles Scribner's Sons, New York, 1906.

Spaeth, R. Ind. Management, lix, 7, 1920.

Taylor, F. W. The Principles of Scientific Management, New York, 1916.

U. S. Pub. Health Service, Pub. Health Bull., 106, Washington, 1920.

——— Pub. Health Rep., xxxvii, 4, 309, 1922.

Vernon, H. M. The Influence of Hours of Work and of Ventilation on Output in Tinplate Manufacture, Industrial Fatigue Research Board Report No. 1, London, 1919.

Webb, Sidney, and Cox, H. The Eight Hours Day, London, 1891.

Weichardt. München. med. Wchnschr., No. 12, li, 1904.

The following publications are of value and general interest and contain extensive bibliographies:

Bainbridge, F. A. The Physiology of Muscular Exercise, Longmans, London, 1919.

Lee, Frederick S. The Human Machine and Industrial Efficiency, Longmans, New York, 1918.

Spaeth, Reynold A. The Problem of Fatigue, Journ. Indust. Hyg., i, 22, 1919.

CHAPTER XXXVI

PNEUMOCONIOSIS

A. J. Lanza

Pneumoconiosis has been attracting a great deal of attention in recent years, particularly in connection with the mining industry, in which it has become associated with tuberculosis to such an alarming degree as to cause national apprehension in many countries.

As commonly met with in clinical practice, pneumoconiosis may be due to the inhalation of silicious dust (hard rock metal mining, quarrying, the pottery industry, etc.), in which case it is termed "silicosis." It may also be due to the inhalation of coal dust, "anthracosis," or to the inhalation of metal particles, "siderosis," or to the organic dusts associated with the textile and other industries. Of all the varieties of this disease, silicosis is by far the most important, both on account of its extent and of its being so closely allied with tuberculosis.

Generally speaking, in regard to inorganic dusts, it may be stated that the harmfulness of any such dust is in direct proportion to the amount of free silica which it contains.

Only very minute particles of dust, 5 microns or less in diameter, can find their way into the lung tissue. The dust particles pass down the air passages into the alveoli, in which they set up a catarrhal process. They penetrate the walls of the alveoli and are carried along the lymphatic channels, causing an inflammatory blocking with the production of fibrous tissue. This fibrous process is at first discrete in character, but may gradually proceed to the complete obliteration of the lung tissue.

The degree and rapidity of development of fibrosis, following the inhalation of dust, depend upon the nature of the dust and the extent of exposure thereto. Anthracosis is a more mild fibrosis than silicosis, while the miner in quartz develops silicosis much more rapidly than the pottery worker. It is, so to speak, a question of dosage.

It might naturally be thought that the fibrosis produced by pneumoconiosis would be a protection against tuberculosis. This is true in the case of coal dust; the exact opposite is true of silicious dust. The reason is not entirely clear. It is supposed that the protective action of coal dust is due to the fact that it stimulates phagocytosis, as a result of which not only the particles of coal dust, but any bacteria that happen to be present, are extruded from the lung. Certainly coal miners are relatively free from tuberculosis, while metal miners have a very high death-rate from that disease, varying in different localities with the nature of the ground in which they work. Recent investigations[1] indicate that the action of silica dust is not, as has been always supposed, mechanical, but largely chemical; and that the hardness, sharpness and insolubility of the silica particles will not furnish the explanation for the extreme fibrosis which they cause, with the consequent liability to tuberculosis.

As far as organic dusts are concerned, their action seems to be mostly limited to irritation of the upper air passages, probably on account of their relatively large particles; nor is their relationship to tuberculosis as definite as in the case of inorganic dusts.

Cement dust, which has been the subject of some dispute, would appear to be similar in action to the organic dusts.

Symptomatology and Prognosis.—The onset of pneumoconiosis is insidious and may extend over years. In a typical case of silicosis, the patient notices a gradually increasing shortness of breath. This may be the only symptom for months and he is not apt to seek medical advice until it causes inconvenience upon exertion. As dyspnea advances a cough develops, which is most troublesome in the early morning, and it is a common complaint of these patients that it causes them to vomit their breakfast. There is little sputum until the later stages of disease or until infection occurs. The patient usually appears quite well and robust, with little or no loss in weight and no cyanosis, even with well-marked dyspnea. As soon, however, as a tuberculous infection becomes activated the clinical picture changes, there is profuse cough and expectoration, afternoon rise of temperature, loss of weight and all the symptoms of a rapidly progressing consumption. A common feature of the earlier stages of silicosis is the tendency to "catch colds."

Physical examination in the early cases may reveal nothing, and even where there is a sufficient degree of fibrosis to interfere with the patient's ability to work, on account of the consequent dyspnea, there may be nothing discernible but weakened breath sounds. A diagnosis is impossible without a knowledge of the patient's occupation and of the hazards incident thereto. The X-ray is of the greatest assistance, revealing in selected cases the presence of fibrosis even before physical signs are present; but the interpretation of X-ray plates of pneumoconiosis is often difficult and misleading. They may resemble tuberculosis so closely that only an expert can differentiate between them. In the later stages there may be dullness on percussion, a number of adventitious sounds may be heard and the rigidity of the chest wall due to the underlying fibrosis is well marked and may call into play all the extraordinary muscles of respiration. Moist râles usually signify infection, generally tuberculosis, but may be absent, due to thickened pleura and dense fibrosis, even when there are tubercle bacilli in the sputum and other symptoms of tuberculosis present. They are nearly always to be heard in the final stages.

---

[1] Mavrogordato for the South African Institute for Medical Research.

It is convenient to divide pneumoconiosis into three stages: (1) where there are symptoms but no disability; (2) where there are both symptoms and an impairment of ability to work; and (3) where there is complete disability. Tuberculous infection may take place at any stage, and practically always terminates the third.

As with tuberculosis "symptoms are more important than signs" and a gradually increasing dyspnea in an otherwise healthy adult who is exposed to a dust hazard in his daily work is strong presumptive evidence of pneumoconiosis, especially if the X-ray shows a bilateral, evenly distributed shadowing.

The prognosis depends upon the extent of the disease and the ability of the patient to change his occupation. Coal miners, while comparatively free from tuberculosis, are prone to bronchitis and pneumonia in later life, but their fibrosis may cause little or no actual disability. In silicosis the prognosis is good if the fibrosis is not extensive and if the patient can secure a dust-free occupation. Otherwise the prognosis is bad and tubercle infection is almost inevitable, particularly where the dust responsible is of a highly silicious nature (60 per cent or more free silica).

Where the patient can secure a more healthful occupation the lung tends to rid itself of dust, and even with a well-marked dyspnea improvement may be looked for.

Treatment and Prevention.—The fundamental requirement in the treatment of pneumoconiosis is that the patient immediately abandon his occupation. In the face of continued exposure to harmful dust there can be no treatment. Otherwise the treatment is hygienic. Both work and sleep in the open air are indicated, and if the disease is well advanced a rest of several months is most advantageous. Unfortunately these patients are all working people, and not only is it often impossible for them to take a rest, but frequently they cannot even change their occupation. In this event, if they can put in the summer working in the fields at fruit picking or other agricultural employment, it will tend to prolong their lives. A sedative cough mixture may be necessary in some cases. In the event of tuberculous infection the treatment does not differ from that of pulmonary tuberculosis in general. It would appear that the families of men who are exposed to silicious dusts do not suffer from tuberculosis to an undue extent. This may be due to the fact that tuberculosis in silicotics usually runs a rapid course, lessening the chances for the spread of infection.

The prevention of pneumoconiosis lies wholly in the prevention of harmful dusts in mines, factories, quarries, stoneworks and the like. The dust must be overcome at the point of origin, either by the use of water, as with the mine drill, stonecrushing, pneumatic stonecutting and the use of grinding wheels, or by the use of a dust exhaust system as seen in factories. Increased ventilation by moving large quantities of air and "diluting" the dust is useful, but will not of itself suffice. Once the dust is in the atmosphere, it is not possible to prevent it being inhaled. Dependence upon respirators is fallacious, for a respirator which would filter out the minute dust particles that cause trouble would offer such resistance to breathing as to render its use impossible for continued work. Mouth breathing increases the susceptibility to dust inhalation, and those subject to this habit, as well as arrested cases of tuberculosis, and those subject to derangements of the respiratory tract, should be warned against working in dusty trades.

REFERENCES

Collis. Silicosis, Milroy Lecture, 1915.

Drinker. Modern Views upon Development of Lung Fibrosis, Journ. Indust. Hyg., Feb., 1922.

General Report of the Miners' Phthisis Prevention Committee, Union of South Africa, 1916.

Landis. Pathology and Clinical Manifestations Following Inhalation of Dust, Journ. Indust. Hyg., July, 1919.

Mavrogordato. Studies in Experimental Silicosis, Report No. 15, South African Inst. Med. Research.

U. S. Bureau of Mines, Reports and Bulletins.

U. S. Public Health Service, Ibid.

CHAPTER XXXVII

OCCUPATION NEUROSES

Clarence A. Patten and Theodore H. Weisenburg

An occupational neurosis, according to Mills, is an affection of the neuromuscular apparatus due to special occupations, coming on after the individual becomes an expert in the particular operation. There are various terms used to denote this condition: fatigue neuroses, occupation spasms, copodyskinesia, anapeiratic paralysis, professional impotence, professional hyperkinesia, and professional dyskinesia. It is found principally in certain types of occupations, particularly those which demand excessive and long-continued use of groups of muscles concerned in highly specialized movements. The following is a partial list: scriveners, telegraphers, stenographers, typists, pianists, violinists, trap drummers, cornet players, seamstresses, sewing-machine operators, artists, dancers, turners, shoemakers, shoefitters, tailors, blacksmiths, carpenters, watchmakers, goldbeaters, weavers, milkers, money counters, compositors, typesetters, grinders, cutlers, cigarette makers, letter sorters, voice users, knitters, etc. From the list given it can be seen that the majority of cases are concerned with identical muscle movements constantly repeated. The individual who works now with one group of muscles, and now another, is seldom affected. It is the person who makes the same muscle movements without variations who develops an occupational paralysis. Further, we find that in many of these muscle movements there is a very limited range; it is often a delicate, precise, patternlike motion such as the telegrapher uses, in which the key is grasped always in a certain manner, moved in a definite manner and with a range of motion less than a half inch in radius. Grosser movements that are patternlike in repetition usually demand a commensurate expenditure of energy, and may become, therefore, similar in effect to those of lesser range.

Particular types of occupation and highly specialized movements are not accountable entirely for the development of this form of neurosis. Overwork and fatigue may be precipitating causes, inasmuch as there is a limit to the amount of reserve in a given muscle and nerve. Crile's experiments in exhaustion show conclusively that under prolonged activity there results—so far as the nervous system is concerned—fatigue of a certain proportion of nerve cells from which they only partially recover, or an exhaustion which does not permit of recovery. In the occupations which require rapid and prolonged repetition of a movement in a limited group of muscles, the centers of nerve control over these groups must be kept in a state of continuous activity. If fatigue affects certain of these cell groups there will develop, as a natural sequence, inadequate muscle response and incoördinate movement. If the nerve centers are overstimulated, the muscles may be thrown into a condition of tonic contraction constituting the so-called "spasms" or "cramps" so frequently encountered. Overstimulation likewise will produce incoördination. Prolonged fatigue or overstimulation will result sooner or later in changes in the higher nerve centers or muscle substance which permanently impair their functions.

Any factor which causes a disturbance of nutrition in the nerve centers or muscles increases the possibility of the occurrence of paralysis, spasm or tremor. Such factors may be found in the toxic products of fatigue, in chronic systemic infections, such as tuberculosis, syphilis, kidney disease, heart disease, etc.; in local disturbances, such as arthritis, tendon affections, bone disease, etc.; and in toxic states from endogenous or exogenous sources, with which every one is familiar.

Probably the most important single consideration is that which concerns the make-up of the individual. Occupation neuroses are most frequently found in those who have a neuropathic constitution, and this, when interpreted broadly, means simply a biological deficiency. The nervous mechanism is limited in resistance and output of energy, and, therefore, when called upon to initiate highly specialized muscular movements, becomes exhausted or damaged. Psychic causes are important in precipitating symptoms, and are based in the same degree on the lack of capacity of the individual—a constitutional deficiency.

The frequency with which occupation neuroses occurs has been given by Thompson and Sinclair of England as 13 out of every 700 to 800 employed. Men appear to be much more frequently affected than women, although there are more women than men employed, according to Osler.

It must be remembered that the locus of toxic influence may be central or peripheral, or both. There are mentioned, among the local physical causes, the effects of continued pressure, continued blows or shocks, varying temperatures, etc. The direct effect of local conditions or central lesions may be similar, manifesting themselves by paresthesias or cramps.

SYMPTOMATOLOGY

Irrespective of the occupation, the symptoms most frequently encountered are spasm or "cramp" of the muscles, sometimes with pain, tremor, paralysis, and incoördination; according to Jelliffe and White, there may be in addition flaccid paralysis, and even athetoid movements. There may be one or several of these symptoms present, but the most frequent combination is paresis and tremor. Of the two sides, the right is most frequently affected, because of the preponderance of dextralities.

The spasms are undoubtedly due to overstimulation. In "writer's cramp," for instance, a flexor spasm, the pen may be held so firmly that it is impossible for a time to remove it (the lock spasm of Weir Mitchell). The cramp may sometimes be so sudden in onset that the pen is hurled from the hand. In telegraphers the extensor spasm jerks the fingers away from the keys. In some instances the spasms are so bizarre that awkward movements of any sort may be produced.

Tremor is a usual accompaniment of the tonic spasms and as frequently is associated with weakness. It is not always confined to the fingers (that is, in scriveners, typists, telegraphers, etc.), but spreads often to the entire arm, and sometimes to the lower extremity of the same side or the opposite side. It is not constantly present, and may be brought out only when the specialized movement is attempted. Once the impression has been made on the suggestible mind of the individual, however, the tremor may be induced by the mere thought of the occupation, or a discussion of his symptoms. It is not extraordinary, then, that later the tremor appears under all sorts of circumstances, since the attention has been fixed on the arm or hand as the weak point; the part may serve the purpose of the individual as a scapegoat for his mental instability. The tremor is of varying degree and amplitude, but often sufficient to suggest a marked incoördination.

True paralysis does not exist. Weakness is frequently the first manifestation, and is usually noted only in connection with the specialized movement—all other motions being normal in every way. It may occur only after the muscular exertion has been continued for a certain period, and possibly, for a time, recovered from by a brief period of rest. Later it is noted at the onset of the movement and disappears only after discontinuance of the act. The patient is conscious in the beginning of a sense of increased effort in writing, then more and more difficulty in holding the pen, until finally he is unable to grasp it at all. Tremor usually accompanies the paresis and gives rise to the apparent incoordination.

Incoördination has been shown in the foregoing to be more apparent than real, accompanying, as it does, the spasm, tremor or paresis.

Pain is rare as such. There is often a disagreeable tired feeling in the muscles, or a sensation not unlike pain during the tonic spasms which has probably given rise to the term "cramp." It is questionable if, when severe shooting pains in the limb are present, an actual neuritis is not the basic condition rather than an occupation neurosis, per se. Frequently the patients complain of various paresthetic phenomena, particularly numbness, a feeling as though the part were asleep, or a tingling in the fingers. Some observers hold that a true neuritis may be a part of the picture, with pain, discomfort, and tenderness on pressure over the nerve trunks, but the chances are that an occupation paresis may develop as a result of a primary neuritis, and not a definite neuritis as a result of the neurosis.

Among other manifestations, not commonly a part of the clinical picture, are to be mentioned vasomotor disturbances. There occur in severe cases a hyperesthesia of the affected part with a glossy skin, and local asphyxia not unlike chilblains. Sometimes on the attempt to perform the specialized function, the part becomes flushed and hot, the veins engorged, and paresthetic symptoms develop. Trophic disorders must be extremely rare, though mention is made in the literature on the subject, of atrophy, hypertrophy and other trophic disturbances. It is commonly held, however, that the possibilities are that one is dealing with a different disease when trophic disorders are found, such as progressive spinal muscular atrophy, cerebrospinal lues, etc. At least, one should be very guarded in diagnosing an occupation neurosis in the presence of atrophy, since the majority of the cases do not show it, and it is not in accord with the usual conception of the etiology and pathology.

The *diagnostic features* then are: a weakness, spasm or tremor of a group of muscles performing a highly specialized type of movement, occurring in individuals who show a predisposition to functional nervous states. Functional and organic nervous diseases must be carefully considered in the differential diagnosis and definitely ruled out.

Functional neuroses are the most frequent conditions presenting symptoms similar to the professional neuroses. While neurasthenic symptoms may be a part of the general picture, the usual features of mental and physical exhaustion—marked irritability, failure of power of concentration, apparent memory loss, hypochondriasis, and sleeplessness—readily differentiate a true neurasthenia. There will be, in clear distinction in the occupational neurosis, a localization of the weakness to some area which is involved in continuous muscle effort.

Hysteria may be confusing, but a careful history, in addition to the manifestations, will usually clear up the diagnosis. The hysteric frequently has a paresis, tremor or contracture of the left arm, less often of the right, and glove or stocking anesthesia is usually present. Definite motivation is likewise found, if searched for, as reason for the hysterical symptoms: a compensation affair, a tendency to dodge responsibility, a wish to attract sympathy or an attempt to get out of a difficult situation. The patient with an occupation neurosis is genuinely concerned because of his disablement and, though neuropathic in predisposition, does not show the marked additional symptomatology. The affected part in an occupational neurosis is normal for all other movements except the one concerned with the occupation.

Psychasthenics may develop an apparent monoparesis, spasm or tremor, etc., in the right arm or hand, but in addition they show morbid mental states characterized by doubts, fears, and compulsions.

The organic nervous diseases—such as amyotrophic lateral sclerosis, progressive muscular atrophy, and syphilitic disease of the anterior horn cells—are characterized by progressive atrophy, paralysis and fibrillary contractions, in muscle groups. There is, in addition, tendency to bilateral involvement, loss of tendon reflexes or spasticity, etc., all of which separate the occupation neuroses from those conditions.

The *prognosis* under any circumstances is rather dubious. If there is a faulty use of the muscle groups in question, a correction of the attitude and use may be sufficient to overcome the disability. Once the condition has developed markedly there is little chance of cure, so far as that type of work is concerned, and the patient would do better in a different occupation. Writers have been taught to use their left hand when the right has been affected, but there has frequently developed the same condition in the left. When it is considered how little the left hand is used, and that, by reason of non-use over a period of development and activity, it is inadequately equipped to pick up the burdens which the right has failed in, it is really not to be expected that permanent or efficient results can be obtained. Add to this also the weakness of the underlying nervous state and the outlook seems exceedingly gloomy.

TREATMENT

Prevention should be the first consideration in the treatment of the occupational neuroses. The types of work which are highly specialized must be carefully studied. There are many "methods" which lend themselves to improvement, and faulty instruments or implements can be altered to suit an individual's requirements. Faulty postures should be corrected and periods of rest instituted sufficiently often to prevent fatigue of muscles. The clearest example lies in the scrivener's work. Here the pen may be grasped too tightly, or a finger movement used in writing. There may be bad desk and lighting arrangement. Substituting a free arm movement for the finger movement in writing is decidedly effective and lessens the possibility of the development of "cramp." Furthermore, the careful selection of employees for specialized work will eliminate many individuals unfit for the work and decrease the possibilities of occupational neurosis.

General measures consist in the elimination of toxic or infectious conditions by internal medication, following the principles usually employed in such states. Physiotherapy, cabinet sweats, hydrotherapy and stimulative electric applications are valuable adjuncts. A proper amount of rest, adequate and nutritious food, and a daily bowel movement are important.

All worry, apprehension and maladjustment in social, economic and domestic life should be corrected. Appreciating the underlying constitutional neuropathy, psychotherapy is indicated. A cheerful and hopeful attitude can be cultivated to good advantage and optimism for recovery and usefulness propagated. Absolute rest from the occupation is necessary.

The neurosis itself may be treated by various mechanical appliances, if the patient depends absolutely upon that particular occupation for his livelihood. There have been devised many ingenious braces and splints for the hand, each adapted to suit the individual's requirements. These, it can be seen, offer but little toward actual recovery and at best are only palliative. Properly, the occupation should be discontinued and the part put at absolute rest. Daily massage and the interrupted galvanic current three times a week aid in restoring tone to the fatigued neuromuscular apparatus. Later, active gymnastic exercises are of great value, particularly those requiring gross movement, and that do not suggest the motion expected in the specialized work. When it is the right hand that is affected, the left can sometimes, and in certain types of work, be educated to perform a similar function, thereby making it possible for the patient to use either hand and prevent fatigue or exhaustion. Frequently it is essentially necessary for the patient to learn a new occupation. The choice of specialized work should be carefully made, avoiding the types which might predispose to recurrences of the neurosis. An occupation which requires motion of wider range and utilizes varying groups of muscles is best suited to such cases.

From the standpoint of drug therapy, nothing is specific. Iron, arsenic, quinin, cod-liver oil and general tonics are valuable in building up the general health. Atropin in 1/100th gr. doses three times a day relieves muscular tension. Of the sedatives, bromids are useful in relieving general nervous irritability and tension. The synthetic drugs, such as veronal, luminal, trional, etc., aid in securing the proper amount of sleep. Narcotics should be avoided in so far as possible, for in this type of case habit formation is relatively easy.

CHAPTER XXXVIII

OCCUPATIONAL DERMATOSES

C. Guy Lane

General Considerations.—In beginning this chapter I wish to emphasize two points which have a bearing on the prophylaxis of this group of diseases. First, *these diseases are occurring with much greater frequency than the average practitioner suspects.* It is probable that 5 per cent, or even more, of all cases of skin disease are caused by factors associated with work which the individual is performing. In those cases where the eruption is limited to exposed surfaces, namely, the hands, arms and face, the percentage of industrial cases would naturally be greater still. It is necessary, therefore, to bear the industrial factor in mind in all cases of skin irritation, and especially any irritation on exposed surfaces. Secondly, *it is of prime importance to realize that detection, that is, the recognition of the industrial factor, is the principal element in the determination of the proper treatment and of future prevention.* A careful inquiry into the substances the patient is handling and the processes in which he is assisting is the duty of all of us in connection with such cases.

There are certain general etiological factors to which it is well worth directing attention briefly. The element of idiosyncrasy or individual susceptibility seems to be a factor of great importance. Theoretically it seems possible that for every substance there are individuals susceptible to this particular substance. Practically it is found that the list of irritants is almost endless, and that frequently only one person out of a large number working at a given process or with a particular substance will have some irritation on the skin due to his occupation. The whole subject of susceptibility is an extremely indefinite one and it is probable that further accurate and careful study of the individual and his environment will eliminate many of the cases from this very indefinite group. We know, for example, that in some trades a dry skin, and in others excessive perspiration, as well as a thin skin, minute trauma, and general cleanliness of the individual, have a share in the causation of these conditions, and there are undoubtedly other factors which have a similar influence. Furthermore, the methods of improper cleansing used by many of the workmen after the work period are frequently a large factor. It is only necessary to mention the traumatizing effect of brushes and sand-soaps, the drying effects of such substances as benzin, gasoline, alcohol, etc., and the effects of various soaps and even of water itself, especially on skins which are naturally dry, to realize that these factors may contribute a share in the causation of this group of skin irritations.

Prophylaxis.—In general the prevention of this group of diseases depends upon keeping the irritating substance from contact with the individual worker. The prophylactic methods used to accomplish this result may be discussed in four groups.

1. *Prevention by Alteration of the Process in Which the Individual Is Participating.*—Directions to a patient to stop work are often impossible to follow for economic reasons. The direction to change his work usually means removing the individual to another department of the factory, shop or store, often with resulting decrease in pay and disturbance to the individual. It should not be forgotten that this same term "change his work" can be applied with better reason to actual alteration or improvement in some way of the process in which he is working in order to prevent future irritation of the same kind in this same individual and in his fellow workmen. This method is especially essential in occupations where large numbers of employees are affected. Various factors combine to make this a less feasible means of prevention than the protection of the individual worker, but the method should not be forgotten.

2. *Prevention by Mechanical Means.*—This group includes the use of gloves of one kind or another; the adoption of various mechanical devices to prevent splashing, and to prevent the circulation of dusts, etc.; the use of clothes fitting tightly at the neck and wrists in the case of exposure to dusts; the use of impervious aprons or trousers in the case of exposure to oil, etc. The individual method will vary with the different occupations and will often be suggested by personal observation of the individual at work.

3. *Prevention by the Application of Some Impervious Substance to Act as a Coating Over the Exposed Areas.*—These parts should be well cleansed with soap and water, and dried, provided the soap does not produce any irritation. This step should be followed by a thorough application of one of the following substances previous to going into the workroom. It is important that the process be repeated before each working period.

a.	Castor oil or olive oil			
	Lanolin		āā	
b.	Acid carbolic	min.	X	(0.6)
	Lanolin	ʒ	III	(90)
	Petroleum jelly	ʒ	IX	(270)

4. *Prevention by a Proper Cleansing Process.*—The detrimental effect of gasoline, benzin, etc., particularly on dry skins, has been referred to. It is hardly necessary to advise against the use of strong soaps, stiff brushes, and much traumatizing in the cleansing process. If one of the impervious substances mentioned above has been applied before going to work, it will be found that the use of a mixture of liquid soap and sawdust, well rubbed in and washed off after the work period is over, will often complete the cleansing process without the application of other chemical or mechanical cleansing.

Treatment.—The aphorism aptly used by Prosser White is well worth emphasizing at this time: "Stop exposure and trade dermatoses heal." It is usually advisable that individuals with the severer types of eruption stop their work until cured, but it is often feasible for those with the less severe types to continue, with the adoption of some prophylactic measure. With the failure of these latter individuals to improve under such methods, even these cases should stop work. Repeated attacks mean the stopping of work, and unless suitable preventive measures can be found it will mean the necessity of giving up the occupation, or the transfer to some other work which precludes exposure to the particular substance to which the individual reacts.

The general principles of treatment, therefore, can be summarized as follows:

1. The detection of the particular factor involved in a given case.
2. The elimination of the offending substance by changing the process, by mechanical prevention, etc.
3. The use of therapeutic measures adapted to the different dermatological manifestations which are produced by industrial factors. These manifestations are discussed in the following paragraphs and for convenience in referring to them have been arranged in alphabetical order.

ACNE

Etiological Factors.—These are dirty or dusty atmosphere, chlorin, oil, tar and other petroleum products. The lesions of this type will therefore be found most frequently in mechanics, in various petroleum workers, among pressmen, in woolen workers, brush workers, chemical workers, etc.

Prophylaxis.—General cleanliness is very essential. Special attention should be given to the proper cleansing of the face with soap and water. Workers should be cautioned against rubbing oily and tarry hands over the face. Proper ventilation should be provided, splashing of oil and chemicals guarded against, and such other mechanical measures adopted as may be indicated by the working conditions.

Treatment.—The fact that these conditions are probably due to plugging of the sebaceous outlets indicates the need for thorough soap and water cleansing, especially upon finishing the day's work. Considerable emphasis should be placed on general hygienic measures, such as bathing, the use of proper diet and laxatives to prevent constipation, etc. A diet low in fat is often advisable. For individuals with an especially oily skin I frequently suggest the use of Tincture of Green Soap, Ranier's natural soap, or the use of one of the more drying soaps, such as Ivory soap. In these cases I advise the use of an alcohol lotion similar to the following, applied at night and washed off with soap and water the following morning:

Sulphur præcip.				
Tincture of camphor	āā	℥	ss	(15)
Alcohol		℥	VIII	(240)

or the familiar lotio alba:

Zinc sulphate				
Potassium sulphuret	āā	ʒ	I-IV	(4-15)
Aqua rosæ		℥	VIII	(240)

In prescribing this lotion it is well to indicate the advisability of dissolving these two substances separately in the rose water and then mixing in order to secure the desired preparation.

Occasionally the use of a peeling ointment seems advisable, such as:

Resorcin	ʒ	II-III	(8-12)
Salicylic acid	ʒ	II-IV	(8-15)
Petrolatum	℥	II	(60)

In the cases in which the condition approaches a real folliculitis black wash is often of use:

Calomel	ʒ	ss	(2)
Aqua calcis	℥	VIII	(240)

ACTINOMYCOSIS

Etiological Factors.—The essential factor is infection by the ray fungus. This disease appears usually in farmers, stablemen, coachmen, or at least individuals who come in contact with animals or straw. It is usually found about the face.

Prophylaxis.—Individuals in the above groups should be sure that their teeth are in good condition and should have any external wounds on the face cared for properly, though infection usually takes place by way of the mouth. The proper treatment of any suspicious lesions in cattle is essential.

Treatment.—It is often advisable to incise the fluctuant areas, express the pus and apply a mild antiseptic wet dressing. These cases will practically always get well with the administration of a saturated solution of potassium iodid, the dose being increased to the point of tolerance of the individual. In my opinion, suberythema filtered doses of X-ray are of distinct benefit in hastening the involution of these lesions.

ANTHRAX

Etiological Factors.—Infection by the anthrax bacillus is the immediate cause. The individuals generally affected are workers among wool, leather workers, furriers, butchers, etc. It is apparently more common from contact with the Chinese and Indian hides and wool.

Prophylaxis.—Quarantine on hides and wool from districts not known to be free from anthrax should be enforced. The sterilization of hides and wool before handling should be of service if it can be done thoroughly and at the same time in a practicable manner. In places where infection is possible, rules should be posted and also enforced for the early treatment of any abrasions on the skin, and also for keeping individuals with skin wounds from working with hides or wool, etc. Early treatment of this disease is imperative.

Treatment.—The expectant treatment, combined with anti-anthrax serum, rather than surgical treatment, offers the best results. Rest in bed, good nursing, antiseptic dressings, such as 50 per cent alcohol, bichlorid 1:4,000 or 1:5,000, saturated boric acid, etc., and the keeping up of the patient's general condition give better results than excision. The anti-anthrax serum should be given locally about the lesion and intravenously. The dosage and frequency of the injections will depend on the severity of the case. In the milder cases 10 c.c. are injected about the lesion every twelve to twenty-four hours, and 40 to 50 c.c. are given intravenously at the same time. Larger doses are given more frequently in the more severe cases.

BURNS

Etiological Factors.—Among these are acids, alkalis, hot liquids and metals, electricity, steam, X-ray and radium. These lesions appear in many varied types of industry of which the following are examples: workers in chemical plants, firemen, molders, employees of electric-light companies, etc., and users of radium and X-ray. During the war there were many burns among ammunition workers.

Prophylaxis.—Carelessness is often a large factor and educational campaigns among employees will often be of service in reducing these casualties. The prevention of splashing, the use of gloves and long-handled tools, together with greater care by the individual worker, are important. The handling of live wires requires the use of suitably insulated gloves and tools as well as extreme care. Effective lead screens and distance are necessary for the protection of radium and X-ray workers.

Treatment.—This chapter is hardly the place for the discussion of the usual first, second and third degree type of burns as commonly seen. In the same way, the treatment of X-ray and radium burns is a subject in itself and the reader is referred to books dealing especially with this subject. Only a few of the more strictly speaking industrial burns will be discussed.

The acid burns are best treated by immediate use of a weak alkaline solution, such as a sodium bicarbonate solution, and following this a 2 to 5 per cent sodium bicarbonate ointment is often all that is necessary.

Alkali burns as a whole are much more serious. In the minor cases a weak solution of acetic acid is applied and a protective ointment, such as boric acid ointment, applied thereafter. The more extensive cases will be deeper and have more necrotic tissue. Chlorinated soda soaks, 1 : 20, and dressings are advisable in clearing these up. If there tends to be delay in healing, some of the stimulative measures given in the treatment of ulcers are advised. In many of the cases, after becoming clean, the use of one of the paraffin dressings, applied while warm with an atomizer or with a brush, often provides excellent protection and allows rapid healing.

Electric burns are apt to be rather deep and obstinate, and after the initial dressing of some substance to exclude the air, such as boric ointment, and morphia if necessary on account of the pain, the above treatment will be advisable to help the necrotic areas separate and stimulate healing. Skin grafting should be used in the extensive cases from any cause.

CALLOSITAS (Callus)

Etiological Factor.—The etiological factor, in this case, is mechanical pressure or friction. It occurs in many occupations at the site of pressure of tools, machines, etc. Calluses are often an index of the type of occupation of the individual. They occur in carpenters, cobblers, leather workers, mechanics, etc.

Prophylaxis.—The prevention of this type of disease depends upon overcoming mechanical factors—the relief of pressure by padding, by gloves, by altering shape of tool or process, etc. As a rule comparatively little can be done by the individual in the line of prevention.

Treatment.—Inasmuch as these calluses are a protective measure developed by the skin, there is seldom call for therapeutic measures except when very marked. Thorough soaking in a hot alkaline solution (sodium bicarbonate ½ to 1½ per cent) will usually soften them so that the thickest part can be easily pared off. In case further treatment is necessary, a 20 per cent salicylated plaster can be applied, or salicylic acid in collodion, a dram to the ounce, painted accurately over the areas and not allowed to flow down on normal skin, can be used two or three times a week.

DERMATITIS (Acute Type)

This disease is characterized by acute onset, redness, vesiculation, oozing, itching, etc.

Etiological Factors.—The dividing line between the cases of dermatitis and of eczema is a rather artificial one and is more for the purpose of grouping them into the acute and chronic forms for proper treatment. The etiological factors in both groups are practically the same and cover a vast number of substances and occupations. It is impossible in a chapter of this sort to give a complete list and only a few such substances and the occupations in which they occur are given: housewives, washerwomen, and allied work, from soaps and washing powders; employees in dye plants, from dyes and chemicals; photographers, from developing chemicals; nurses, from formalin, bichlorid, etc.; silver polishers, from cyanid; rubber workers; candy factory employees; tannery workers; shoe workers; painters; machinists; packers (using straw, etc.); bakers; cement workers; tobacco workers; wool workers; printers, etc.

Prophylaxis.—In the acute cases it is best to stop work absolutely to prevent extension of the process and the exacerbations which are apt to occur if the individual continues at work.

In both the acute and chronic types the prevention of future attacks lies in a most careful study of the individual himself, of the substances with which he is working, and the processes in which he takes part. It is not enough to say that the eruption on the individual is due to working in a rubber factory, for example. An attempt should be made to ascertain as far as possible the exact substances causing the eruption. Then directions should be given for the use of such preventive measures as may be indicated in accordance with the suggestions made in the paragraph on General Prophylaxis.

Treatment.—For the proper treatment of an acute dermatitis of any severity due to occupational factors I repeat that it is advisable to have the individual give up his work temporarily. This prevents continued exposure to the irritant or irritants, and provides for a rest of the affected parts from the irritation of continual motion. To relieve the itching, lessen the oozing, and to act as a mild astringent, some one of the well-known whitewashes is advisable.

a.	Carbolic acid	min.	XX-XXX	(1-2)
	Zinc oxid	ʒ	III	(12)
	Calamine	ʒ	I	(4)
	Aqua calcis	℥	VIII	(240)

To the above lotion may be added menthol, gtt. 5 to 15 (0.3 to 1 gm.), as an additional antipruritic, or if this lotion seems too drying, glycerin, drams 1 to 3 (4 to 12 gm.) may be added.

b.	Talc				
	Starch	āā	ʒ	VI	(25)
	Glycerin		ʒ	V	(20)
	Aq. saturn.				
	Aq. dest	āā	℥	IIIss	(100)

Occasionally in some of these acute conditions I use the so-called alum acetate solution, either full strength or diluted down to a 25 per cent solution:

c.	Alum	ʒ	ss	(2.5)
	Lead acetate	ʒ	III	(12.5)
	Aqua	℥	VIII	(250)

I have also found that a saturated boric solution works very well in these conditions, either alone or as a diluent of one of the above lotions.

Weak alkaline lotions, sodium bicarbonate, .5 per cent, or ½ to 2 per cent solution of sodium hyposulphite, may also prove of value. As the acute symptoms fade away with the resultant scaling, roughened, dry skin, it is advisable to use, perhaps in conjunction with the lotion, some of the milder ointments suggested in the Treatment of Eczema.

ECZEMA

Under this title are included the comparatively chronic dermatitis cases characterized by chronicity, redness, infiltration, fissuring, itching, and perhaps oozing and crusts. This includes many cases in which daily or at least frequent contact with a minute amount of a certain substance will produce such a chronic form rather than the acute type mentioned above. Many cases with acute onset will not be seen by the physician until this stage is reached.

Etiological Factors.—Some of these factors have been discussed under Dermatitis and attention is also called to the general etiological factors mentioned in the first part of this chapter.

Prophylaxis.—See discussion under Dermatitis.

Treatment.—In the treatment of these cases it is necessary (1) to relieve the itching in order to avoid discomfort to the patient and the necessity for scratching with its consequent trauma; (2) to stop the oozing; (3) to lessen infiltration and aid the return of the skin to normal condition. It is often best to begin the treatment of these cases with soothing applications. For this purpose the lotions mentioned in the treatment of Dermatitis are often excellent. In these it is frequently advisable to use slightly more glycerin or to add the menthol as suggested.

A modified Lassar's paste, applied twice a day, is often very soothing:

Zinc oxid	ʒ	ss	(2)
Amyli	ʒ	IV	(15)
Petrolatum	℥	I	(30)

If a stiffer paste is needed, the starch may be increased to one ounce in this prescription. Menthol, gr. 2 to 5 (0.1 to 0.3 gm.), may be added to this paste if necessary to relieve the itching.

In some of these cases another form of paste is of service:

Salicylic acid	gr.	V	(0.3)
Bismuth subnitrate	ʒ	I	(4)
Amyli	ʒ	II-III	(8-12)
Ung. aq. rosæ	℥	I	(30)

or a smooth oily preparation such as the following:

Boric acid	ʒ	I	(4)
Bismuth subnitrate	ʒ	ss	(2)
Olive oil	ʒ	II	(8)
Lanolin	ʒ	VI	(24)

perhaps with menthol, gr. 2 to 5 (0.1 to 0.3 gm.), added.

In the more chronic obstinate cases it is well to investigate the general condition of the individual in regard to constipation, indigestion, diet, possible contact with other irritants, the action of soap and water, etc., for the possible effect which these factors would have on the skin condition. In these cases no one remedy will be found satisfactory and a frequent change of agents may be necessary. Such changes, however, should be made cautiously. Crude coal-tar, ½ to 1 dram (1 to 2 gm.) to the ounce of the modified Lassar's paste, is often most beneficial. One is often surprised at the way in which the crude coal-tar paste will relieve the itching and hasten the return of the skin to normal. It is a most valuable adjunct in the treatment of eczema. I believe that naftalan will act in much the same way, though my experience with this agent is limited. In some of these cases the stimulation provided by the oil of cade, min. 5 to 20 (0.3 to 1.3 gm.), in the same paste or the use of ichthyol, dram ½ to 1½ (1 to 3 gm.) will be of service. Diachylon ointment is another agent of much value, particularly in the dry, thickened, slightly itching hands as they gradually return to normal.

In numerous cases I have seen the X-ray, unfiltered, in one-eighth to one-quarter skin-unit doses at weekly intervals relieve the itching and hasten the process of involution.

Many other agents are used in various types of eczema and not infrequently one must experiment with various preparations in a given case before finding a satisfactory one.

EPITHELIOMA (Skin Cancer)

Etiological Factors.—These are sun, exposure to weather, continued irritation by such substances as tar, pitch, soot, paraffin, X-ray and radium. These conditions are frequently seen in farmers, sailors, paraffin workers, shale oil workers, pitch and tar workers, and radium and X-ray workers.

Prophylaxis.—Routine body washing and cleanliness in some of these occupations; proper clothing and protective measures to prevent the action of sun and contact with substances causing affection; special protective measures for individuals working with X-ray and radium are demanded. Employees should be encouraged to report for the examination of any persistent, though apparently, minor lesion.

Treatment.—It is essential that treatment of these lesions be begun at the earliest possible opportunity. The treatment consists in complete eradication by: (1) surgical removal; (2) X-ray or radium in sufficient amount to cause destruction; or (3) by curetting or caustics in *competent* hands.

FOLLICULITIS

Etiological Factors.—The most important etiological factors are oil, paraffin, pitch and tar. It appears, therefore, in much the type of workers listed in the paragraph on Acne.

Prophylaxis.—Proper cleanliness of the individual is important, and frequent changing and boiling of underclothes is necessary to prevent a continuance of the condition. Before entering the workroom a thorough rubbing in of some one of the impervious substances recommended under general prophylaxis is very important. This should be followed by the thorough cleansing with the liquid soap and sawdust mixture recommended in the same paragraph. Furthermore, the adoption of necessary mechanical measures, including the use of impervious trousers or aprons, is recommended.

Treatment.—Frequent cleansing baths are indicated. General immersion in baths of boric acid solution are of value in the generalized type. Itching occurs in many of these cases, is often intense and it is frequently advisable to use one of the antipruritic lotions advised in the section on Dermatitis.

Black wash, either full strength or diluted, has also been of great service:

Calomel	ʒ ss	(2)
Aqua calcis	℥ VIII	(240)

The following ointment has proved of marked value in these cases:

Salicylic acid		
Sulphur præcip.	aa ʒ I	(4)
Petrolatum alba	℥ II	(60)

This ointment should not be used immediately after using the black wash.

The following is often of use:

Ichthyol	ʒ ss-II	(2-8)
Petrolatum	℥ I	(30)

In some cases the use of unfiltered X-ray in doses of one-eighth to one-quarter skin unit at weekly intervals has been of distinct value. In many of these cases it has seemed as if stannoxyl internally, gr. 5 (0.3 gm.) t.i.d., has been of use. I do not feel that vaccines have assisted the progress of these cases.

KELOIDS

Etiological Factors.—Among these are burns, especially with acids, trauma causing almost any sort of break in the skin, operations for industrial accidents, etc.

Prophylaxis.—No successful method of prevention of keloids is known except such measures as tend to prevent the occurrence of the etiological factors.

Treatment.—For large keloids, in locations favorable for operation, the most successful treatment is excision, followed by deep, filtered radiotherapy. The majority of the other lesions, particularly the younger, more rapidly growing type, respond rather readily to filtered radium or X-ray. Suberythema or erythema doses, filtered through 1 to 3 mm.

of aluminum, at monthly intervals, usually result in marked improvement. The earlier the treatment is begun the better the response of the growth.

MALINGERING (Dermatitis Factitia and Artificial Eruption)

This condition is included because malingering is found in all occupations and is often extremely difficult to distinguish from the real skin irritations due to occupational factors.

Prophylaxis.—There is no prophylaxis. It is possible, however, that closer medical supervision and examination, particularly of new employees, may aid in the eradication of this class of cases.

Treatment.—In cases where there is a suspicion that the lesions are artificially produced or deliberately maltreated, it is well to provide for dressings only by the physician or nurse, and applied in such manner that it will be apparent if the dressing has been disturbed. It is usually necessary to keep these cases under observation until absolute proof is obtained. Then an attempt should be made to correct the fundamental neurotic condition, usually in coöperation with a neurologist. Treatment of the individual lesions will vary according to the type of lesions produced and the agents used.

PARONYCHIA

Etiological Factors.—The continual or frequent immersion of the hands in soap and water or other cleansing agents is the principal factor, that is, among individuals doing housework, among dishwashers, soda clerks, etc.

Prophylaxis.—Prophylactic measures in these individuals are of little use. Rubber gloves do not prove satisfactory and it is impossible to use any of the impervious substances indicated earlier in this chapter. It is usually necessary to give up the type of work the individual is doing.

Treatment.—It is usually advisable to have the individual discontinue any work involving the keeping of his hands in soap and water. The use of mild antiseptic dressings is of service in the less chronic type: potassium permanganate, 1:4,000 to 1:8,000, or the use of the alum acetate solution given above. Black wash is often a very valuable form of treatment.

Calomel	ʒ ss	(2)
Aqua calcis	℥ VIII	(240)

An ichthyol ointment

Ichthyol	ʒ ss-II	(2-8)
Petrolatum	℥ I	(30)

is oftentimes of more service in the chronic type. I have also found the following ointment of value in some cases:

Acid salicylic		
Sulphur præcip.	aa ʒ ss	(2)
Petrolatum	℥ I	(30)

Fractional doses of unfiltered X-ray have been used in some cases with good results. Ultraviolet light has been used with some success in the treatment of these cases. The condition is often extremely rebellious to any form of treatment.

PUS INFECTIONS

Etiological Factors.—Most important are infections with staphylococci and streptococci. These conditions usually appear as a complication of acne, folliculitis, dermatitis, etc., and following slight trauma of various kinds. They appear as abscesses, furunculosis, infected wounds and scratch marks, cellulitis, etc.

Prophylaxis.—The preventive measures for certain of the above conditions have already been discussed. To these methods should be added the prevention of trauma by the use of gloves, long sleeves, machine guards, etc., and the prompt and proper treatment of small wounds and abrasions.

Treatment.—In the superficial and impetiginous infections, boric acid soaks and dressings, using a saturated solution, followed by an ointment of ammoniated mercury, gr. 10 to 30 (.6 to 2 gm.), in boric acid ointment often will be sufficient. In some of these cases a chlorinated soda solution, 1 : 20 to 1 : 40, is very useful when used in the form of soaks and also as wet dressings. Dakin's solution can also be used. Ichthyol in ointment, from 5 to 25 per cent, is of value in some cases, though I personally have not found this substance of as great value as many reports would indicate.

In abscesses and cellulitis the usual surgical treatment—incision, drainage and hot wet dressings—is indicated. In the cases of generalized boils—furunculosis—general cleanliness is essential, and the utmost care must be taken to prevent continual reinfection. The sterilization of underclothing and the use of general body soaks in boric acid or weak bichlorid solutions are usually indicated. Incision and drainage and frequent dressings to prevent the spreading of infection are necessary.

Stannoxyl internally, gr. 5 (0.3 gm.) t.i.d., I find of great benefit, and occasionally yeast will prove of value. The patient's general condition, and especially the examination of the urine, must not be forgotten.

TINEA (Ringworm)

Etiological Factors.—The essential element is infection with a fungus, usually belonging to the trichophyton group. The disease occurs in handlers of horses and cattle, that is, among stablemen, farmers, etc.

Prophylaxis.—The proper treatment of irritated areas appearing on skins of these animals is necessary, as well as greater cleanliness on the part of individuals coming in contact with them.

Treatment.—It is necessary to pull out the hairs in the diseased area or produce epilation by X-ray if the disease occurs in a hairy area. On the glabrous surfaces and after the epilation has been produced, one of the following antiparasitic ointments is indicated:

a.	Sulphur sublim.			
	Phenol	āā	ʒ I	(4)
	Naphthol		ʒ ss	(2)
	Adipis		℥ I	(30)
b.	Hydrarg. ammoniat.		ʒ ss-I	(0.2-4)
	Ung. zinc oxid		℥ I	(30)
c.	Acid salicylic			
	Sulphur præcip.	aa	ʒ I	(4)
	Petrolatum		℥ I	(30)

Tincture of iodin can be used daily for four or five days, combined with manual epilation, but the use of this substance and of any strong antiseptic should be avoided within ten to fourteen days of any X-ray treatment.

TUBERCULOSIS OF THE SKIN

Etiological Factors.—The tubercle bacillus is the great etiological factor. These conditions occur among butchers handling infected meat, among physicians, among nurses and attendants of tuberculosis hospitals, among postmortem room attendants, etc.

Prophylaxis.—Strict inspection and the elimination of all infected meat; greater care and cleanliness on the part of individuals handling suspected meat; the use of gloves and careful asepsis in tuberculosis sanitaria and in postmortem rooms; all these are important.

Treatment.—Many of these lesions will respond very readily to X-ray treatment, an erythema dose being given with filter once every three or four weeks. It may be necessary to curet thoroughly or to excise a certain number of these lesions. In any case the general health of the individual should receive proper attention.

ULCERS

Etiological Factors.—These are chrome, paraffin and pitch, acids and alkalis. The condition may be found in chrome workers, chrome tanners, wool dyers, etc., chemical workers, lime burners, etc.

Prophylaxis.—It is necessary that care be taken mechanically to prevent splashing of these substances so far as possible. Very frequent rinsing of hands and arms is advisable. Gloves are hardly of real service in any of these occupations. In some cases it may be possible to use some one of the impervious substances suggested in the previous section. A weak solution of bisulphite of soda has been suggested to counteract the effects of the chromic acid and chrome compounds if used immediately after exposure.

Treatment.—These lesions should be kept covered and be dressed daily, or at least every second day. Chlorinated soda solution, 1:16 or 1:20, is a valuable adjunct in cleaning the ulcer of sluggish tissue. Dakin's solution, either used as a solution or in the form of a paste, will often be found of value. When the ulcer has become clean, the use of some mild protective ointment may be sufficient to allow the lesion to heal.

Many of these lesions are very sluggish and some sort of stimulation will be necessary. Balsam of Peru will aid in some cases. Scarlet red, in 5 per cent ointment, is one of the best. It should be applied to the edge of the ulcer every other day, applying boric acid ointment on the alternate days. The scarlet red should be stopped at the least sign of irritation of the surrounding skin. The ultraviolet light is often of marked service in stimulating epithelial growth. It is well to start treatment at twelve-inch distance with the air-cooled lamp, for two, three, or four minutes, depending on the extent and type of lesion and the character of the individual's skin. This can be repeated at four to seven-day intervals, gradually increasing the exposure.

TROPICAL DISEASES

CHAPTER XXXIX

INTRODUCTION—THE RÔLE OF THE MEDICAL MAN IN THE FUTURE CONTROL OF THE TROPICS

WILLIAM B. WHERRY

Encircling the earth, between 30° N. and 30° S. of the equator, are tropical and subtropical regions—the most beautiful, the most fertile, the most richly endowed portions of the globe. Time and again they have been invaded by northern races in search of wealth. Stricken by strange pestilences of mysterious origin, the invaders disappeared. Gradually the rumor spread and the belief became ingrained that there lay "the white man's grave." One hundred years ago Dr. James Johnson wrote:

"Whoever has attentively examined the posterity of DeGama, and Albuquerque, now scattered over the coast of Malabar, the plains of Bengal, and the island of Macao, once the theaters of Lusitanian preëminence, will be tempted to exclaim:

" 'Twas not the sires of such as these
Who dared the elements and pathless seas;
Who made proud Asian monarchs feel
How weak their gold 'gainst Europe's steel,
But beings of *another mold*—
Rough, hardy, vigorous, manly, bold!"

Will racial decadence be the fate of those whites who are now contemplating an expansion into the tropics in the near future? Will modern science, operating through the medical man, be able to neutralize the forces which act deleteriously upon the white man in the tropics? Upon the solution of these questions must rest the white man's future relation to the tropics.

Leaving out of consideration the results of the old spirit of discovery, conquest, and religious zeal, the effects of which upon the present ownership of the tropics are scarcely perceptible, there is one factor above all others which has instigated the seizure of tropical possessions by modern white races. With the overcrowding of the temperate zones and ultimate scarcity of food ever before them, these peoples have sought to provide room for expansion. That the tropics already have been largely appropriated may be seen from the following table, prepared for Sir Wm. Osler by Mr. H. O. Becket of the Department of Geography, Oxford, England. It shows at a glance the tropical possessions of the four western nations —England, France, Germany, and the United States—in 1909.

TROPICAL POSSESSIONS OF ENGLAND, FRANCE, GERMANY AND THE UNITED STATES IN 1909

Nation	Tropical Territory in				Total Tropical	Home Country
	America	Africa	Asia	Pacific		
France	***440,000**	**17,700,000**	**18,500,000**	**80,000**	**36,720,000**	**39,000,000**
	35,000	4,032,000	310,000	9,000	4,386,000	207,000
German Empire	Nil.	**11,700,000**	Nil.	**400,000**	**12,100,000**	**60,000,000**
		931,500		96,000	1,027,500	209,000
United Kingdom	**2,000,000**	**30,500,000**	**296,600,000**	**1,150,000**	**330,250,000**	**44,500,000**
	109,000	1,600,000	1,900,000	1,400,000	5,009,000	121,000
United States.	**305,000**	Nil.	Nil.	**7,707,000**	**8,012,000**	**76,000,000**
	47,500			134,500	182,000	2,970,000

* Heavy figures give population. Lighter figures give area (in square miles).

However, it is extremely uncertain that these possessions will serve for the purpose of expansion. With characteristic audacity the white man is attempting to accomplish in the near future that which has resulted from the operation of natural laws acting through thousands of years—the acclimatization of an alien race.

It is true that the problems facing white settlement in the tropics a comparatively few years ago have been greatly modified by recent advances in our knowledge of tropical diseases, but these researches tend rather to promote the success of the temporary settlement of white labor in the tropics and to increase the efficiency and supply of the black labor of the tropics than to guarantee their successful and permanent settlement by whites.

Formerly it was held that heat, the use of peculiar foods, the abuse of alcohol, sexual excesses, the lack or abuse of exercise, etc., were the factors which lowered the resistance of the white exotic transplanted to an unaccustomed environment. Recent researches have convinced many that most of the damage was due to protozoön parasites—the Entamebæ, the Plasmodidæ, the Leishmaniæ, the Trypanosomidæ, and metazoön parasites, such as Uncinaria, Clonorchis endemicus, Opisthorchis noverca, Schistosoma japonica, Porocephalus armillatus, etc. It is quite possible for hygiene to protect the white man from these dangers; but, granted that this has been accomplished, can he be insured immunity from the injurious effects of tropical sunlight?

According to present belief, the white man to become climatically naturalized must accomplish two things: he must acquire more pigment in his skin and go for the greater part unclothed, and, in addition, readjust his thermoregulatory mechanism—nervous and cutaneous. This can never be accomplished excepting through the operation of the slow and uncertain laws of natural selection.

Those who lay special stress on the significance of the pigment in hides and hairs of geographically distributed animals call attention to the fact that the pigment in the skin of all races is the same, and the amount present in each is at least sufficient to exclude the dangerous rays of sunlight encountered in the particular latitude of the particular race home. Hence, the blackest skins are found in unforested Africa, the plains of India; yellowish and brownish skins in jungle tribes or on the humid coastlands in the tropics, or in the desiccating grass lands in intemperate climates; the whitest skins in high latitudes with moist atmosphere and cloudy skies. Similarly, among the lower animals are cited, as extreme examples of adaptation, the zebra and the polar bear.

However, there seems to be no proof that the actinic rays in tropical sunlight exert more than a superficial effect on man. It has not even been demonstrated that the sunlight contains rays of sufficient penetrating power to reach the pigment deposited in the deeper layers of the skin. Nor is there proof that it contains a greater proportion of ultraviolet rays; there is only proof, to date, of its greater intensity. Freer and Bacon have shown that the chemical activity of the sun's rays is from 5 to 20 times greater in Manila in October, November, and February than in Chicago during the months of May and June. They encountered great variations in its activity, for example, two successive, equally clear days were actinic and non-actinic respectively. The destructive action of sunlight on many microörganisms is established, and it is known that plants cannot survive geographic changes without the elaboration of sufficient chlorophyll of the type necessary to absorb light of particular wave-lengths, but it does not follow that these arguments can be applied unhesitatingly to the action of light upon higher organisms. Yet, on the basis that pigment is developed according to need, it has been advised that the white man settling in the tropics must dress in clothing which is opaque to light or of a color sufficiently near the lower end of the spectrum to exclude the dangerous rays. The results of such protection have been conflicting and generally unsatisfactory; but this question needs to be reinvestigated, for, according to Freer, recent work shows that color is immaterial as long as it presents a considerable absorption band in the proper region of the spectrum.

The other side of the argument, namely, that the injurious effects of the tropics on whites is due to the intense heat combined with excessive moisture, seems to have been more definitely established. Recently Aron has brought forward very convincing experiments showing that the heat-producing rays exert a more injurious effect than the actinic upon animals naturally provided with a heat-regulating mechanism. The rapid, deadly effect of tropical sunlight on certain mammals could be obviated by measures which promoted loss of body heat, for example, loss through the action of an electric fan. He further showed that brown skins, which should theoretically absorb more heat in sunlight, show less rise in temperature in tropical sunlight than the skins of whites under similar conditions. This difference is attributed, and it would seem correctly so, to the earlier and more rapid loss of heat by water of evaporation. So that the main difference, in their relative resistance to a tropical climate, between naturalized and alien races lies chiefly, if not entirely, in the delicacy and efficiency with which cutaneous heat regulation is performed. Evidently those measures promoting heat loss, for example, going uncovered as much as possible, protection from sunlight, frequent bathing, the liberal consumption of cooling, non-alcoholic drinks, etc., are only secondary in importance to the prevention and eradication of infectious diseases. In short, as Johnson's motto expresses it, we must "study well the clime, mold to its manners our obsequious frames, and mitigate those ills we cannot shun."

However, putting aside for the present the question whether the white man can permanently acclimate himself in the tropics, the fact remains that the tropics are largely in his possession, and that he will have to face the problem of developing their enormous natural wealth as a source of supply for an overcrowded world—not by the old reprehensible system of exploitation, but by holding and developing them as a trust for civilization.

It is amazing to what extent the English-speaking world is already dependent upon the production of the tropics. Benjamin Kidd gives the following instructive figures:

1896—United Kingdom trade with the tropics	£138,000,000
1895—United States trade with the tropics	70,000,000
Total	£208,000,000
Combined trade of the United Kingdom and United States with the remainder of the world outside of English-speaking lands	473,000,000

The importance of the tropics to the English-speaking world as a whole is further made apparent when one considers that the trade of the United Kingdom and that of the United States with the tropics in 1896 and 1895 respectively amounted to some 38 per cent and 65 per cent, respectively, of their total trade with the remainder of the world.

It is not likely that the inhabitants of the temperate zones will grow less dependent on imports from the tropics, unless scientific agriculture develops a system of intensive cultivation undreamed of at present. Noone believes that the natural resources of the tropics will be developed by native races—as witness Brazil and Central America. An adequate supply can be obtained only by the use of temporary white labor or by native labor under the supervision of a temporary white population. The world-wide efforts which are being made to make the tropics habitable for whites will insure the success of temporary settlements of white labor. These have been shown to be economical in Manila, in the Panama canal zone, in the Queensland sugar plantations, etc. Further, by removing the parasitic factors in negro lethargy, they will increase the efficiency of black labor; by decreasing mortality they will increase the supply of this black labor. So that the study of tropical diseases is not only of scientific but also of economic interest.

For further details concerning hygienic victories in the tropics, the works of the late Sir Rupert Boyce and that of Sir Ronald Ross are especially recommended; and for details concerning the influences of geographic environment the reader is referred to the researches of Ellen Churchill Semple.

REFERENCES

Aron, H. Investigation on the Action of the Tropical Sun on Men and Animals, Philippine Journ. Sc., Series B, vi, 101, 1911.

Boyce, R. W. The Mosquito or Man? 1909.

——— Yellow Fever and Its Prevention, 1911.

Freer, P. C. The Tropical Sunlight, Philippine Journ. Sc., Series B, v, 1, 1910.

Kidd, B. The Control of the Tropics, 1898.

Lyde, L. W. Climate and Racial Skin Color, Contemp. Rev., London, xcix, 231, 1911.

MacDonald, T. F. Tropical Lands and White Races, Journ. Trop. Med. & Hyg., ii, 139, 1908.

Osler, W. The Nation and the Tropics, Oxford University Press, London, 1909.

Phalen, J. M. An Experiment with Orange-Red Underwear, Philippine Journ. Sc., Series B, v, 525, 1910.

Ross, R. The Prevention of Malaria, 1910.

Semple, Ellen C. Influences of Geographic Environment on the Basis of Ratzel's System of Anthropo-Geography, 1911.

Woodruff, C. E. The Effect of Tropical Light on White Men, 1905.

CHAPTER XL

INSOLATION

Paul G. Woolley

Insolation (siriasis, sunstroke, heat stroke, thermic fever) is a term applied to certain symptom-complexes that are the result of disturbances of heat regulation. These disturbances arise primarily from physical causes—the heat and water content of the air about the body, the condition of the peripheral vascular circulation, etc.—but these causes lead to or produce chemical changes in the organism which are manifested in the characteristic clinical symptoms.

According to the sequence of the symptoms, that is, according to their rapidity of development and their severity, various classifications have been made in the group of insolation. The simplest is that which includes heat exhaustion and sunstroke. The former refers to syncope from exhaustion "caused either by the direct rays of the sun or a heated atmosphere in the shade." The latter refers to the results of "the general heating of the whole body, blood, and tissues, which may happen either from prolonged exposure to the sun's rays in a heated atmosphere, to a heated atmosphere in the shade, or to an abnormal thermotaxic state due to vasomotor or other heat-regulating disturbances."

From this statement it appears that there is little justification for a classification other than a much simpler one which would arrange the cases into mild, severe, and peracute. The causes of all are obviously the same. The symptoms are phases of the effects of heat upon bodies which differ in one or another direction or degree. The type and amount of variation is determined by many factors—the use of alcohol, overeating, overwork and fatigue, bad ventilation, anxiety, worry, personal uncleanliness, wearing of too tight or too heavy clothes, and others.

An epitome of this argument is found in a recent article to the effect that heat exhaustion is produced by high temperature; heat exhaustion brought on by exposure to the sun is sunstroke.

Insolation has always been attributed to exposure to the sunlight, and more particularly to exposure of the head to the direct rays. While this is true, in part, it is not true of all cases, and it does not account at all for the large group of border-line cases that are classed as heat exhaustion or prostration, which are encountered among stokers, laundry workers, and persons who are crowded together or who live in hot, badly ventilated, insanitary rooms, buildings, or barracks. Taking the whole series of cases that may be included in the group (represented by the classical hyperpyrexial sunstroke, by atypical apyrexial heat stroke, and by heat prostrations which occur in the absence of direct sunlight), we may assert that insolation may occur under any circumstances in which heat accumulates in the body, and in which the body does not rid itself of its metabolic end products, notably perhaps acids. And, since metabolism increases under the influence of abnormal heat, it will appear that symptoms of insolation are imminent whenever the heat production of the body exceeds the heat dissipation.

Hirsch believed that, while heat was the primary factor, the immediate cause of trouble was a diminution of oxygen and retention of toxic principles. Vincent also stated his belief in a toxic basis. Gordon says that recent work tends to the conception of the paralyzing action on the nervous system of some toxic element which produces metabolic changes in the neurons, and, according to the degree of auto-intoxication, the effect of the sunstroke will be either an attack of ordinary heat prostration or syncope with unconsciousness or death. If this be true, then insolation is an auto-intoxication brought about by substances formed in the body under abnormal conditions of heat retention.

How the bodily heat regulation is upset we do not know, but, from the fact that heat accelerates chemical reaction, and from the fact that the symptoms of sunstroke are also largely the symptoms of shock, we may surmise that there is some chemical point common to the two conditions. The only probable common factor that occurs to one is the increased acid content of the tissues. In sunstroke this may be the result of increased katabolic chemical activity under the influence of heat. However this may be, we are told that the alkalinity of the blood is decreased and that the oxygen content of the body is diminished.

It is, however, with the problem of solar rays in their relation to insolation that workers have busied themselves, and it is work upon these problems that has given most insight into the question of the production of insolation.

For a time men were led to suppose that the violet and ultraviolet part of the spectrum was most active. This supposition was based upon the results of experiments on the effect of ultraviolet rays upon bacteria, protozoa, and plants, which showed beyond question that these lower forms of life were seriously affected by these rays. It has, however, been shown that the results reached with organisms that have no heat-regulating apparatus do not apply to other organisms that have such an apparatus. Also, Freer, Gibbs, and Bacon have shown that the tropical sunlight

(Manila) contains no more ultraviolet rays that does the light of temperate zones (Chicago). And yet in tropical climates heat prostrations are more numerous (in troops especially) than in temperate climates. Experiments with underwear which prevents the action of ultraviolet rays on the body have also been unproductive of good results. More interesting still, however, are observations such as those of Chamberlain, which render it very doubtful that the actinic rays play an important part in tropical deterioration of any sort.

In the absence of any direct evidence of the ill effects of the ultraviolet rays, the attention of Aron was turned to the red and infra-red rays, with the result that very definite and useful information has been developed.

The heat rays may be active in one of two ways, or in both. They may act by increasing the temperature of the air, or by heating the body itself. Either action tends to decrease the heat dissipation from the body, or to increase the heat accumulation in the body. Aron's work indicates that both ways are important.

The body loses heat in three ways—by conduction, by radiation, and by evaporation. Under normal circumstances radiation is the most active, but, as the surrounding temperature rises, radiation becomes necessarily less important and evaporation (perspiration) becomes more active, until at temperatures which equal or exceed that of the body evaporation alone is of value. Vierordt estimated the normal heat losses through various channels as follows: the urine and feces, 1.8 per cent; by expired air, 3.5 per cent; by vaporization of water from the lungs, 27.2 per cent; by evaporation from the skin, 14.2 per cent; by radiation and conduction, 73.0 per cent. Rubner showed that the heat losses, estimated in calories, by radiation and conduction, took place according to the following table:

HEAT LOSSES BY RADIATION AND CONDUCTION

Temperature	Calories Lost by Radiation	Calories Lost by Evaporation
7° C.	78.5	7.9
15° C.	55.3	7.7
20° C.	45.3	10.6
25° C.	41.0	13.2
30° C.	33.2	23.0

These figures indicate that under normal circumstances radiation is the most important method of physical regulation, but that under abnormal circumstances evaporation becomes the most important. Without increased evaporation the body would not merely retain its heat, but would absorb heat from its surroundings. This state of things had led Hill to remark that, so long as evaporation is active, high temperatures alone will not change the temperature of the body. It is only when evaporation is insufficient that heat produces serious results.

This is shown in a remarkably clear way by Aron's experiments with dogs, cats, guinea pigs, and rabbits, in all of which evaporation takes place only through the respiratory tract. Aron shows first of all that dead animals, in which there is no possibility of heat regulation of any sort, absorb heat very rapidly when exposed to heat, either in the shade or in the direct sunlight, and that heat accumulates far more rapidly in the sunlight than in the shade. He shows, also, that, when in living animals heat dissipation is diminished by reducing the possibility of evaporation (by tracheotomy), the temperature of the animals rises much more rapidly than in normal animals, but less rapidly than in dead animals, while at the same time control animals, which have also been tracheotomized, but which are kept under otherwise normal conditions, live comfortably and show no abnormal variation of temperature and no symptoms due to heat retention.

In a series of experiments with monkeys Aron showed that these animals, which are as accustomed to tropical climate as any that can be obtained, when exposed to the direct sunlight, quickly died, though the control animals kept in the shade, even that of an umbrella, survived. But he also showed that monkeys kept in the sun, but in a current of air, such as that produced by an electric fan or a natural breeze, suffered no damage. He further showed that, if monkeys were placed in an apparatus so that their heads were exposed to direct sunlight, but with their bodies protected and kept cool, they suffered no damage. He concludes, therefore, that it is not the direct effects of the sunlight which do damage, but the hyperthermia produced by the heat rays. He believes that the heat radiated from the sun warms the body tissues more rapidly than can be compensated for by the regulatory mechanism of the body. The tissues and the blood increase in temperature to a point higher than is compatible with life. Apparently the organs most susceptible to this increased heat effect are the brain and heart, though it is not indicated from the lesions in these organs which are most essential in causing death. These characteristic lesions are hemorrhagic lesions in the brain and (in monkeys) in the heart.

Not less clear than the results of his experiments with animals are Aron's results with human beings. They indicate, as he says, "that the temperature of the human skin increases in the sun, but does not reach the normal body temperature. In animals without sweat glands the skin temperature rises above febrile heights, and the tissues lying underneath are heated. On the other hand, penetration of heat through the human skin seems improbable, the effect of the rays being neutralized by water evaporation on the skin. The more perfect this water evaporation is, the better the normal body temperature may be maintained." "Monkeys exposed to the sun in Manila die in little over an hour because of their limited capacity to evaporate water, while man, with his well-developed sweat glands, resists the same climatic condition for a much longer period without detriment." The great importance of this evaporation is shown by the remarkable fact that a man lying quietly in the sun lost 280 gm. in weight in 1 hour. The above figure does not include the water held in the man's clothes, which was also actually lost to the body. The volume of respired air in such a man increased 23 per cent.

The foregoing facts account very well for the usual cases of sunstroke. There remains, however, a small group of cases in which but slight increase of temperature has been observed. Aron accounts for these upon the basis of sudden loss of large quantities of water. He quotes P. Schmidt, who observed, under unfavorable climatic conditions during muscular work, water losses as great as 1 kg. per hour. He proceeds to call attention to the fact that, since the body cannot withstand a loss of water of more than 10 per cent of its weight, an acute loss of from 3 to 4 kg. must necessarily result in grave disturbances, collapse, or death. In this connection it is to be remembered that large losses of water may occur with little reduction of the body temperature, as, for instance, when the sweat runs down and off the body "in streams." Where losses of water are so great, evaporation is reduced, and refrigeration decreased. Serious results may, therefore, result from paralysis of the vasodilators on the one hand, or by too great loss of water from the sweat glands on the other. "It is not the sweat we see, but that which we do *not see,* which exerts the cooling effect."

One further point of interest (among many others) is taken from Aron's work, and this has to do with the question of the exposure of the human head to heat. Aron states that the temperature of the air in the thick black hair of a Filipino may reach 50.1° in an hour of exposure.

Prophylaxis.—If the foregoing facts are true, then prophylaxis consists in preventing the accumulation of abnormal amounts of heat in the body. To accomplish this there are mediate and immediate methods. The former apply to habits and clothing—in general to hygienic surroundings at any time; the latter to habits during severe hot weather.

To enable oneself to withstand great heat the regulatory system of the body should be kept in the normal state of adaptability to circumstances. The skin should be kept clean to allow free evaporation; the clothes should be loose and light, moderately absorbent, but thin enough to allow the air to circulate freely, and so assist evaporation. The diet should be one that will not create unnecessary demands upon the system. Alcoholic drinks should be tabooed, plenty of cool water or fruit juices may be indulged in to advantage, since they serve to keep the excretory system freely open and to encourage refrigeration by evaporation. Fatigue, worry, and anxiety should be avoided. Heavy work in the open should not be done, and even in the shade should be limited. Dwelling houses and living rooms should be open, the windows shaded in such a way as to exclude the heat rays without cutting off what breezes blow. Electric fans or punkahs are often indispensable, and cool (not cold) baths are infinitely refreshing as well as useful in keeping the body at ease.

During times of intense heat, and always during the hot hours of the day, all physical effort should be reduced to an absolute minimum. At such times rest in the shade, with as little clothing as circumstances permit, and, if possible, where even the slightest breezes blow, should be freely indulged in. Under such circumstances electric fans, when possible, and punkahs, where they are not, are not only pleasant, but almost necessary. The oriental custom of taking a siesta in the middle of the day is a highly commendable one which Europeans should be counseled to follow. In addition to these peaceful recommendations, it is advisable to suggest a light diet accompanied by as much water as can be gratefully consumed. Such a régime will result in quiet of mind and body and will encourage a useful, moderate degree of perspiration and evaporation, which will result in a comparatively mild state of refrigeration. Attention should be paid to the excretory organs. Citrous drinks encourage the renal activities, and so assist in the cleansing of the tissues. Much water will assist intestinal elimination and so assist in preventing intoxication from fecal stasis. Constipation must be avoided with as great care as diarrhea. Any exposure to the direct sunlight should be avoided as much as possible, and when such exposure is necessary it should be brief. An umbrella is a valuable protection.

Treatment.—The treatment of insolation is rather an unsatisfactory matter. This must be so because of the grave changes that are produced in the various organs of the body by the action of the intense heat and the products of abnormally rapid (and very probably aberrant) metabolism. One cannot expect to remove extravasations of blood in the brain, meninges, or heart in a few minutes or hours, and still less can one expect to restore the damage produced by the hemorrhages in less than a very considerable time. Moreover, the disease is rapid. Its victims fall where there are no therapeutic resources. They must be carried to some place where remedies are at hand; where ice can be had, where baths can be given.

There are two main objects in view in treating any form of insolation: to reduce the heat content of the body and bring about elimination of its toxic contents. When these things have been done, *tempus edax rerum* alone will attend to the hemorrhages, the destruction of tissue, the fatty degeneration, and the rest. But even time will not always allow the body to return to its former state. Often residual symptoms remain, such as a tendency to relapses, to headache, to partial loss of memory, to loss of sustained attention, to polyuria, or even to glycosuria. There is the further possibility that insolation, particularly the moderately severe type, predisposes to meningitis.

Treatment of an attack of insolation is, therefore, one that combines refrigeration with elimination, both active enough to produce good effects without embarrassing organs, such as the heart, which are already damaged. To accomplish the former the practice is to give ice-cold packs or baths. To compass the latter, recourse is commonly had to stimulants. This seems not to be the best, except as a last resort. Stimulation of an already burdened organ can do little good and much damage. Hydrotherapeutic methods are of more value in insolation than any drugs. Ice-packs or iced baths have a great value, but should not be continued for more than a few minutes, one to two (Chandler) at a time, and should be discontinued when the rectal temperature has reached 104° F. To replace the water lost to the body before the attack, and to increase elimination, there is no better method than infusion of saline solutions. If it be true that the oxygen content of the body is low and the acid content high, then such solutions should be alkaline. Such alkaline solutions as those recommended by Fischer are extremely efficacious, whether given by rectum or intravenously, in neutralizing the acids of the body and increasing water elimination by the kidneys. The solution for rectal use should be prepared as follows:

Sodium chlorid	10 gm.
Sodium carbonate ($Na_2CO_3.10\ H_2O$)	10 gm.
Water	1,000 c.c.

The injection should be given slowly enough to allow retention. The time consumed in injecting a liter should not be less than one hour.

For intravenous injection the following solution may be used:[1]

Sodium chlorid	14 gm.
Sodium carbonate (crystallized)	10 gm.
Water	1,000 c.c.

This also should be given very slowly. The effect of these solutions upon the secretion of urine is remarkable, and, as a rule, they will make it unnecessary to use digitalis. When this latter drug is used, it should be very carefully administered and its effects carefully watched. The use of strychnia is not advised in the active stage of the disease.

Such treatment will dispose of the immediate danger, and when this has been done treatment is symptomatic. Headache may be treated with acetanilid, phenacetin, salicylates. But these drugs should be used with extreme caution. This is particularly true of the coal-tar products. Sleeplessness may be controlled with bromids, trional, or other mild sedatives and soporifics.

---

[1] For intravenous use the alkaline saline solution should be very carefully prepared. The carbonate cannot be boiled. The salt solution should therefore be made and sterilized, after which the crystals of carbonate may be added. *If the crystallized sodium carbonate is not available and use must be made of the ordinary dried form of the salt, the amount indicated in the above prescription should be divided by 3.*

In apyrexial heat exhaustion external hydrotherapeutic measures are uncalled for, and treatment should be eliminative and stimulant. The internal hydrotherapeutic methods should be very useful in these cases and should be combined with friction, massage, and warm packs with sufficient internal stimulant medication.

After recovery from an attack of insolation great caution must be observed by the patient to prevent recurrences from subsequent exposures to heat. He must pay careful attention to prophylactic measures, and use the greatest precautions in ordering his life. It is advisable for all such persons to arrange their time in such a way that they may spend the hot seasons at some high altitude or cool summer resort. Many tropical countries have what is known as a "summer capital," to which many people go to escape the heat of the lowlands.

REFERENCES

Aron. Philippine Journ. Sc., Sec. B, vi, 101, 1911.
Bacon. Quoted by Freer.
Chamberlain. Philippine Journ. Sc., Sec. B, vi, 427, 1911.
Chamberlain and Vedder. Ibid., Sec. B, vi, 383, 1911.
Chandler. Quoted from Gordon.
Fayrer. Allbutt and Rolleston's System of Medicine, 494, 1896.
Fischer. Nephritis, New York, 1912.
Freer. Philippine Journ. Sc., Sec. B, v, 1, 1910.
Gibbs. Quoted by Freer.
Gordon. Osler's Modern Medicine, i, 52, 1907.
Hill. Quoted by Aron.
Hirsch. Quoted by Gordon.
Hubner. Quoted in Howell's Textbook of Physiology, 913, 1910.
Maude. Quoted by Chamberlain.
Phalen and Nichols. Philippine Journ. Sc., Sec. B, vi, 525, 1911.
Vierordt. Quoted in Howell's Textbook of Physiology, 913, 1910.
Vincent. Quoted by Gordon.
Woodruff. The Effects of Tropical Sunlight in White Men, New York, 1905.

CHAPTER XLI

TROPICAL INFECTIONS OF BACTERIAL OR UNKNOWN ETIOLOGY

Samuel T. Darling, Richard P. Strong and Paul C. Woolley

UNDULANT FEVER (MALTA FEVER)

Samuel T. Darling

This endemic fever is derived from goat's milk infected with Micrococcus melitensis, a pleomorphic microörganism which, as was pointed out by Evans, is closely related to the bacillus of infectious abortion of cattle. These two organisms are related serologically and are now known as Brucella, B. melitensis being regarded by some as a substrain of B. abortus.

The disease may be regarded as a chronic septicemia, for cases of the fever last from sixty to one hundred and fifty or more days. The microorganism is found in the peripheral blood for very long periods (one to two years) and is in large numbers in the spleen. Bassett Smith has observed a secondary anemia to follow the disease with a low polynuclear count and increased lymphocytes. Localized swellings and necrosis of hepatic cells occur, with round cell deposits and a toxic affection of the peripheral nerves from which a chronic neuritis supervenes. Severe cases are complicated by hemorrhages and purpura. The spleen becomes enormously enlarged. The organism is eliminated from the urine and feces. Light cases may be mistaken for malaria. There is severe frontal headache, irritability, restlessness, insomnia, acute sciatic pains and tender spleen. A typhoidal state often supervenes.

B. paramelitensis is more pyogenic than B. melitensis and cases of suppuration and orchitis are apt to be due to the former. Hence vaccines prepared with B. paramelitensis should be used in chronic cases showing these types of infection.

While the disease is endemic in the Mediterranean basin, it has been detected occasionally and sporadically in distant lands. Craig reported a case in the United States in 1905. Later cases were reported in soldiers returning from the Philippines. Ferenbaugh reported cases from Texas, and Lake believes the disease has existed in Texas and Arizona and the southwestern part of the United States for years. Most of the cases have occurred among people who have used or been concerned in handling goat's milk. In Europe fresh goat's milk and cheese freshened in goat's milk have been incriminated.

The disease is diagnosed by agglutination tests, the organism being agglutinated in dilutions up to 1 : 2,000 in chronic cases.

Treatment

Serum and Vaccine Treatment.—Trambusti and Donzello have used a specific serum with success in treating children.

Vaccine therapy has been used with some degree of success by Bassett-Smith and others. Its efficacy depends on the ability of vaccines to raise the defensive powers of the body. Sensitized vaccines when administered early in the disease have shortened the duration of the fever to eight weeks. Vaccines also ameliorate the various symptoms, whether local or general, and they prevent the occurrence of relapse. No bad symptoms follow their use and they may be given subcutaneously, intramuscularly or intravenously. Durian de Cottes, who has had a wide experience in treating undulant fever, uses vaccines when the temperature is falling, and during the intervals of apyrexia. The dose is made proportional to the degree of fever, but in an inverse degree. An initial dose of from 25,000,000 to 50,000,000 is given, rarely going beyond 100,000,000. The earlier the vaccine is administered in the disease the better, hence the importance of early diagnosis. Vaccine treatment should be continued once begun, and in cautiously increasing doses, reducing them before a febrile accession and continuing until the patient is immunized. The doses of vaccine are given in series of eight or ten with an interval of ten to fifteen days between the series. In ambulatory cases and those only moderately severe, the vaccine may be regularly but cautiously used to avoid severe reactions. Vaccines are contra-indicated in cases in which cardiac or renal lesions existed prior to the attack, and also in cases of hyperpyrexia and hemorrhage. Durian prepares vaccines with the aid of chloroform instead of heat, believing that the resultant reactions are milder and without any loss of efficacy. This observer advises the use of serotherapy and chemotherapy in hyperpyrexia and in the acute phases of the disease.

Colloidal therapy has been introduced in the expectation of obtaining a direct bactericidal action and beneficial results have been reported by Ziemann, who treated cases with one or two intravenous injections of from 2 to 4 c.c. of a 2 per cent preparation of collargol. Souleyre advises against the use of colloidal preparations in very toxic cases. He believes it best to increase the dose gradually from day to day, beginning with

1 c.c. and doubling the dose on successive injections, guarding against the depressive effects of the medicament by an injection of oil of camphor or of adrenalin shortly before each dose of the colloidal preparation. Colloidal therapy seems to have a place in the long-standing moderately severe cases.

Symptomatic Treatment.—In a chronic disease of this character careful treatment and nursing are required, the various symptoms being dealt with as they appear. The resourcefulness of the physician and the resistance and patience of the patient are sorely tried in the prolonged siege usually borne, for the disease is not unlike typhoid with several intercurrent relapses. The measures applicable in typhoid fever will be suitable here. The fever when severe is to be treated by means of the cold sponge bath or ice-pack; moderate degrees of fever with tepid sponging. This will tend to alleviate the nervous symptoms. The joint pains will require hot fomentations or the application of dry heat. Morphin should be avoided as much as possible for fear of inducing habit. The milder degrees of pain may be relieved with aspirin or phenacetin. Insomnia is an annoying symptom and opium in some form may have to be given when bromids or either veronal or trional fail. The skin and bladder must be carefully watched. Liquid diet during the febrile stage is enjoined. During the intermission of fever, a light but nutritious diet should be given. Meat diet should be restricted. During convalescence a decided change of scene is beneficial.

It is difficult to recognize undulant fever early but, as soon as it is suspected, the patient's dejecta should be thoroughly disinfected as in typhoid fever. The patient is to be regarded as a carrier and his movements regulated. The microörganism may live for long periods in dust and in soil. Goats and their milk are the chief agencies in the spread of the disease and the sale of goat's milk should be prohibited unless thoroughly pasteurized. The importation of goats from epizoötic regions should be prohibited. Infected goats are to be sacrificed. It has been shown that the disease may be prevented by the subcutaneous injection of killed cultures of the specific microörganism.

DENGUE AND DENGUE-LIKE FEVERS

Paul C. Woolley

Revised by Samuel T. Darling

Dengue is a specific non-contagious exanthematous fever of unknown etiology. It is characterized by its sudden onset, which is marked by headache; by intense aching pains in the joints, muscles, in the back and in the bones; by the appearance of an inconstant transient initial erythematous rash; and by the development of a high temperature. After a few days of fever and often extreme, aching discomfort, a remission occurs which is followed by a second rise of temperature, accompanied by pains, and a more constant polymorphous eruption.

When dengue appears in a community it usually occurs in the form of an epidemic, which spreads with the greatest rapidity and which attacks a very large proportion of the population in a very short time. In this respect it is very similar to influenza, to which it has been supposed by some writers to bear a very close relation. The prognosis is, however, generally good. The mortality never exceeds 1 per cent, and is usually lower than that. Old and infirm persons, those weakened by other diseases, and the young children are more seriously affected than healthy adults, and it is among these classes of individuals that the greatest number of deaths occur. Complications are rare.

Dengue, though it occasionally appears in temperate climates—witness the epidemics of Charleston, S. C., Savannah, Ga., and Philadelphia and the recent epidemic of 1922 when from May to December it prevailed from Texas to South Carolina and Arkansas, visiting every Gulf State, and in proportion to their coast line and Stegomyia index—is a disease of the tropics and subtropics. It is a disease of hot seasons, and epidemics die out with the appearance of cool weather. It is localized to low-lying, seacoast districts, and is apparently not influenced by the moisture of the air. In all of these respects it resembles yellow fever, which disease it may also occasionally simulate in its symptomatology. It is not contagious, and is no respecter of race, nationality, age, or sex.

An interesting feature of this disease is its polymorphic character and resemblance to other more serious diseases. In different parts of the tropics it is called "six-day" and "seven-day" fever. When it appeared on the Isthmus of Panama in 1912 it was called "dengue" in Colon and "six-day fever" in Panama.

The characteristic qualities of dengue—its sudden onset, its rapid attack upon all parts of the organism, its rapid epidemic progress—point to its infectious origin and to the probability that its cause is present in the blood-stream at a very early stage of the disease. And so most of the efforts to discover the cause have been concentrated upon the blood. Hare says that Hunt observed certain motile bodies in fresh blood and in cultures made from the blood. Graham and Eberle also thought they had discovered an organism in the red blood-cells of dengue patients. But these observations have not been confirmed, though evidence has been adduced to prove that the cause is certainly present in the blood, although it is not made visible even by the highest powers of the microscope. The investigations of Graham in Beirut and of Ashburn and Craig in the Philippines have shown that in those localities the disease is spread by a domestic mosquito Culex fatigans (quinquefaciatus). The last-named observers found that the virus was resident in the blood and was a filter passer. Couvy reports that in an epidemic in Syria in the direct examination of the blood he found a short slender spirochete before the onset of the fever.

Cleland and Bradley in Australia were not able to transmit dengue by means of Culex fatigans but they were able to transmit the disease with Stegomyia calopus (Aëdes egyptus) fed on dengue patients during the first two days of the disease.

The experiments of Craig indicate that four days and eighteen hours elapse between the time of infection by means of mosquitoes and the onset of symptoms, and that the virus is present in the blood for from three to four days after the beginning of symptoms.

These experimental facts indicate further similarities between dengue and yellow fever, and indeed pappataci. The most interesting difference seems to be that there is no evidence of a developmental cycle of the dengue organism in the body of the mosquito; in other words, that the infected mosquito is immediately able to transmit dengue, whereas in yellow fever twelve days, and in pappataci five to seven days, must elapse after feeding.

Prophylaxis.—In the Philippines and in the Canal Zone it was found that the disease did not spread within a ward room provided the patients were protected by screening. The disease which had existed for some time in Corrigidor Island at the entrance of Manila Bay was entirely wiped out by obliteration of mosquito-breeding places and by the use of screening protection. A rational prophylaxis then should consist in the reduction in the numbers of domestic mosquitoes Stegomyia and Culex fatigans. The disease will not spread from the littoral for any very great distance inland, excepting along large rivers. The patients should be placed under a mosquito bar or within screened rooms at once. The quarters of the patient should be fumigated with pyrethrum or sulphur. The breeding places of Stegomyia should be reduced to a minimum and fish placed in water containers to destroy the larvæ. Careful policing of the district should be done to remove and destroy breeding containers, and to inspect for possible breeding places.

Treatment.—The treatment, which is purely symptomatic, should be directed towards relieving pain, insomnia and anorexia, reducing the fever and cheering the patients up during the period of depression which occurs in some cases.

Most patients are sick enough to go to bed and all should be required to do so and under a bed net if possible. The pains in the joints and the backache are frequently very severe, even excruciating, and are increased on walking. In addition to the insomnia there is a great deal of restlessness and resultant discomfort due to changing position in bed. Joints which have been the seat of disease or injury are often exceedingly painful.

A preliminary purge of calomel followed by a saline laxative should be given in all cases. The fever during the paroxysms may be reduced by means of tepid sponging. Pains of a moderate degree of intensity may be relieved with milder drugs, such as phenacetin gr. v every three or four hours with an ice-cap for the headache. When they are severe morphin sulph. from 1/8 to 1/4 gr. will be required. Joint pains may be relieved by external heat. For the insomnia and restlessness potassium bromid xx to xxx gr. should be given in water before sleep is desired. The weaker or milder sedatives should be tried at first.

Anorexia is usually severe even in the mildest cases, and it is better until the temperature is normal to give no solid food at all, and but just enough fluids to relieve thirst. Epigastric distress frequently follows the attempt to take solid food. Vomiting and nausea may be controlled by means of cracked ice and such aërated water as is fancied by the patient, sipped slowly, well iced. After the fever has gone it is inadvisable to take solid food for a few days. The stomach, as in yellow fever, is mildly affected. In dengue as in yellow fever there is considerable mental depression during convalescence. Patients are at "zero hour" for a few days and require to be cheered up and told that the slump will be over in a day or two. This depression is possibly more acutely severe than in any other disease. A complete change of scene is beneficial and desirable in these cases. An excellent tonic is the elixir of iron, quinin and strychnin taken before meals.

PAPPATACI FEVER

Samuel T. Darling

Pappataci fever is an acute specific fever of very short duration lasting only about from fifty to sixty hours. The dominant symptoms are referable to the nervous system, rheumatoid pains in the body, an erythematous flushing of the face after defervescence in half the cases and disturbances of the gastro-intestinal tract, epigastric pain and diarrhea. The access of fever is quite sudden, the temperature rising to 102° F.

The disease is known as three-day fever, phlebotomus fever and sandfly fever, and is spread by the fly Phlebotomus papatasii. The etiological factor is unknown. Described originally from the Mediterranean littoral it has since been found in South Africa, South America and India (Chitral).

Treatment.—The fever is mild in character and is treated symptomatically. The patient should be put to bed and given a saline cathartic.

During the access of fever there is physical weakness and general distress, as well as vertigo, and these symptoms are relieved by quiet, preferably in a darkened room. The fever can be reduced by means of a tepid sponge bath and this will relieve the discomfort due to the hot dry skin. Pains may be alleviated to some extent by aspirin in doses of 5 to 15 gr. t. i. d. There is anorexia and cough, for which respectively a milk diet and some sedative or expectorant mixture may be employed. When convalescence is prolonged a change of scene is beneficial.

Prophylaxis.—The fly is too small to be kept out of quarters or barracks by means of ordinary screening. Efforts should be directed against the fly in the larval state in the breeding places. These should be drained or filled. Grass cutting for two or three hundred yards around quarters is an excellent way of keeping these and similar pests out of the sleeping quarters. Fumigants and smudges are sometimes used, but they are usually ineffective.

TSUTSUGAMUSHI FEVER

Samuel T. Darling

A typhuslike fever is endemic in certain parts of the Orient, as along flood plains of Japanese rivers, in Korea, the Philippines, and in recently cleared jungle in Sumatra and the Malay Peninsula.

The fever supervenes upon the bite of one or more species of minute arthropods. In Japan the species incriminated is Microtrombidium akamushi. In Sumatra two kinds of mites and possibly ticks have been presumptively incriminated of spreading the disease. A small ulcer, covered with a crust, usually is found at the point of attachment of the mite. The lymph-nodes draining the ulcer are enlarged. These two features are essential characters of the disease.

A rash appears towards the end of the first week of fever. This is roseolous and maculopapular and spreads over most of the body. The febrile reaction is very much like that of typhoid fever; in fact Schuffner calls the disease "pseudotyphus." The fever falls, however, by lysis or remits after the fourteenth day. Relapses are noted as occurring in some cases. Nervous symptoms dominate the picture, the headache being severe, and there is drowsiness, great restlessness and delirium.

The mortality varies from 3 per cent in Sumatra to 30 per cent in Japan.

Prophylaxis.—The disease resembles Rocky Mountain fever and typhus. From the fact that it is spread by mites which carry the unknown infectious agent from the natural reservoirs—field rodents and other wild and domestic animals in the river valleys, fertile plains or jungle margins—these places must be avoided, particularly in the seasons when the mites are numerous and the disease is rife.

Treatment.—There is no specific treatment and recovered blood does not tend to destroy the virus in vitro. Neither quinin nor arsenic has proved of any value in treating the disease. The therapeutic indications are essentially those of typhoid fever.

SPRUE

Samuel T. Darling

Sprue is a disease of warm climates; however, cases not infrequently are introduced into the United States in persons who have lived several years abroad, and a number of cases have been reported in persons living in the South and occasionally in other parts of the country.

The term "sprue" appears to have been derived from a Dutch word used for that form of stomatitis known in English as "thrush" or "parasitic stomatitis." In British India the disease is known also as "diarrhea alba" because of the color of the stools. Van der Burg called the disease which he studied in Batavia, Java, "Indische spruw." The term would seem to imply that the Indian "spruw" or thrush was a severe or fatal type of the thrush found in adults in the Orient. This fact possesses relevancy in view of the belief held by some that tropical sprue is caused by a fungus, Monilia (Oïdium, Saccharomyces, Endomyces).

Thrush of children, it must be remembered, is a disease that is associated with the presence of Oïdium albicans but which does not arise in a normal mucosa and is found after improper food and in cachectic states, and after a prolonged siege of fever. It is also found in well-nourished, as well as emaciated enfeebled children suffering from diarrhea. It will appear that there is a great deal of analogy between thrush of children and its parasite and the sprue or "Indische spruw" of adults and the parasite associated with it.

There is still some lack of agreement as to whether sprue is a specific malady. Rogers has stated that it is merely a clinical term and there may be, as in dysentery, more than one causative agent. Musgrave, in 1902, felt that sprue was a condition which might occur in various intestinal infections. According to Manson, a great clinical authority, the condition is characterized by a chronic catarrhal inflammation of portions of the alimentary tract. The tongue and mouth are usually inflamed and the mucosa eroded. There is flatulent dyspepsia and pale, phenomenally copious stools which are generally loose, frothy and fermenting. Great tendency to relapse with alternating periods of quiescence and exacerbation of the symptoms is noted. Wasting and anemia are often extremely severe. Manson recognized that the symptoms might arise as a primary condition, or supervene on other affections of the bowels. Generally he thought there was a disturbance of the biliary function of the liver and probably of the glandular organs of digestion. Slowly progressive, it would terminate seriously in atrophy of the intestinal mucosa if not treated properly. The disease has been confounded with pellagra. The chief endemic foci are Southern China, Malaya, Indonesia, Ceylon, India and the West Indies. Cases have been reported from the Southern United States.

Etiology.—Predisposing causes are prolonged residence in the endemic area, a circumstance which is not infrequently followed by a condition of exhaustion or at least of enfeeblement. Manson believed exhausting diseases, syphilis, frequent childbearing, miscarriage and uterine hemorrhage to be important contributory causes. Bad food and water, anxiety and alcoholism predispose to the disease. While Manson did not feel that malaria predisposed to the condition, this infection was thought to inaugurate the symptoms in two cases seen in the Malay peninsula.

The affection is more common among those who are foreigners to the endemic region and who have come from more northerly lands. Europeans and half-castes, Chinese and Bengali's who have emigrated to more tropical climates, have a higher attack rate than natives.

Kohlbrugge and Manson-Bahr believe that a Monilia is the cause of the disease; Ashford that Monilia colonizes in the alimentary canal when there has been a preceding syndrome caused by a digestive glandular deficiency.

Mayo Robson found that in many cases diagnosed as sprue there was really an inflammatory disturbance of the pancreas, and in others some pancreatic involvement. Brown, in a small series of cases from Baltimore, found the pancreatic ferments to be absent or nearly so from the stools and duodenal contents. Michel says "it is strongly evident that the Monilia psilosis of Ashford is the etiological factor in sprue. Low and Castellani believe that the Monilia infection is secondary.

Pathology.—The pathology has not yet been thoroughly studied. There is emaciation and general atrophy, which is particularly noticeable in the liver. The intestinal wall in some cases is thinned out and atrophied as in pellagra, although this atrophy may be a part of the general atrophy seen in this condition. Slight superficial ulceration occurs here and there in the small or large intestines, sometimes in the esophagus. The bone marrow in certain cases has presented a very remarkable pathological appearance. Many bizarre forms of cells were seen, indicating that the marrow was exhausted. Bahr found thinned-out mucosa with tenacious mucus containing mycelium. A round-cell deposit has been described beneath the epithelium with atrophy of the villi.

And in one of my autopsies there was a similar round-cell proliferation change in the uvula.

Symptoms.—The tongue is angry and red with superficial ulcers. The inflammatory change in the tongue and esophagus provokes severe pain on taking such customary foods as curries or even light wines, or other diluted alcoholic drinks. One of the first signs of the disease is the inability to take curry without discomfort. The inflammatory changes in the bowels and alimentary tract bring about peculiar changes in the character of the stools and in their number. The stools are frothy, copious and of a peculiar grayish color, due to pathological change in the bile constituents occurring in the colon, and to an increase in the fat content over normal limits. Examined under the microscope many gas bubbles may be seen. Scrapings from the ulcers of the intestinal tract do not always reveal Monilia; those from the tongue always do and other microörganisms with some frequency such as forms resembling Nocardia and allied fungi.

Prognosis.—The prognosis is very bad in persons over fifty years of age.

Treatment.—This should be preventive, for in spite of our ignorance there is testimony that the disease undoubtedly supervenes on exhausted and enfeebled states brought about in various ways. Few tropical communities were better cared for—in the way of freedom from debilitating disease and in being provided with abundant and nutritious food, pure water and most excellent hygienic surroundings—than the Canal employees in Panama and not a single case of sprue occurred among them. Pellagra did appear among the uncared for ex-employees beyond the pale and living out in the bush. Treatment must be given early and it must be sustained and thorough. In the severely exhausted stages it is hopeless. All underlying pathological conditions which can be recognized, such as dysentery or chronic untreated malaria, should be remedied. Attention was called above to the marantic states in infants upon which thrush is superimposed, improper food, cachectic states, etc. Roux and Linossier found that the parasite of thrush could not be cultured in saliva and this circumstance leads to the view that thrush flourishes in infants before the salivary secretion is well established and to the likelihood of sprue being due in some way to a diminution in the salivary secretion, caused by various debilitating diseases or other pathological conditions. Patients from cooler climates should be sent away from the tropics to remain away.

Symptomatic treatment.—This is begun with the administration of a saline aperient. The patient is kept at rest. British writers in India recommend that special attention be given to clothing and that it be warm enough to prevent chilling. Various diets have been used, such as the milk, milk and fruit, fruit, meat and milk, and meat diets. Milk is given in small quantities at a time. It may be aërated with an alkaline water and either sipped or taken with a tube or sick feeder; limewater should be added if there is much constipation. At first about 5 pints per diem as a total quantity should be taken, in ten or twelve meals increasing to 6 or 7 pints. According to Manson, six weeks after mouth irritation has ceased, and stools have become solid, the patient may sit up and a semi-solid diet may be given. Eggs cooked or beaten up with milk, fruit, and some of the malted cereal foods may be taken. If more nourishing food is needed, evaporated milk may be added to the dietary. Dried subacid fruits may be prepared in various ways. Meat juices and fatless pounded and minced meat and chicken, cooked by roasting or steaming, may be given. Fermented milks may be used to vary the monotony of the fresh form. Castellani recommends the use of soup prepared from sheep or calves' liver in the milder cases of sprue after the return to ordinary food. Alcohol must be interdicted.

Manson used to advise the use of arseniate of iron and, while Rogers states that arsenic should not be administered to patients with sprue, the condition of the bone marrow would appear to warrant its use where there is severe anemia. Brown, finding the relative absence of pancreatic ferments in sprue patients, advises treatment with large doses of pancreatin or pancreatic extract, from 5 to 10 gr., with from 20 to 40 gr. of calcium carbonate or lactate, three times a day, two hours after the larger feedings. This was in addition to general measures such as absolute rest, fresh air and sunshine and a selected diet. Cases with subacidity or anacidity were given acid hydrochlor dil. Improvement was rapid but the pancreatic extract had to be kept up to avoid a return of symptoms.

Castellani, who has had extensive experience treating this rather formidable disease, has recently recommended as a routine treatment for sprue: rest in bed on a milk diet; treatment of the sore mouth by means of an alum-carbolic mouth wash. Injections of emetin are given and large doses of sodium bicarbonate are given by mouth as well as intravenously. More rapid improvement has followed than by the dietetic treatment alone. Acute diarrhea may be checked by means of opium and lead acetate or by the opium and chalk mixture.

Begg recommended the use of yellow santonin but this has not been found to possess any merit in the hands of others who have tried it in cases of the disease.

Specific treatment.—A variety of organisms has been found in the lesions in sprue. Rogers and Nicholls prepared a vaccine from oral streptococci taken from the lesions, and they reported results which to them seemed encouraging. Cultures were made on glycerin agar from sore tongues and vaccines made 100,000,000 to the cubic centimeter. An initial dose of ½ c.c. was given weekly, increasing to 2 c.c. Ashford and Michel and others report favorably on the results of the use of autolyzed vaccines of Monilia psilosis. Autogenous cultures are shaken in a machine for two hours and heated to 37° C. for five days. They are then sterilized at 56° and 0.5 per cent phenol is added. A standard vaccine contains 1 per cent of sediment by centrifugalization. The first injection is 0.5 c.c. This amount is increased gradually to 1.4 c.c. at intervals of from eight to fourteen days. The first injection causes aggravation of the soreness of the tongue and of the gastro-intestinal symptoms. A gain in weight is a notable feature of the beneficial results of treatment.

Hill Diarrhea

This form of diarrhea is seen in the tropics in the cooler regions of the hill stations and other localities in the higher altitudes. From the frequent morning stools of a frothy character with which the patients suffer, the affection has been regarded by some as a variety of sprue, but the tongue is not affected as in that disease.

The etiology is unknown. The affection manifests itself when after residence in the warm lower altitudes the person removes to 3,000 feet or more above sea level. Abdominal pain is the first symptom, usually in the early morning hours, and relief is experienced after expulsion of each stool of which there are several, large and frothy in character. The feces are grayish, due to some pathological change in the bile constituents. There is flatulence and the abdomen is distended. No particular weakness is experienced and the person is quite able to go about. The symptoms recur, however, each morning, and in some persons particularly susceptible the affection may become chronic and take on a serious aspect. This is expressed in the occurrence of flatulent dyspepsia, distaste for food, and disinclination to work. Removal to a location at a distinctly lower altitude, as at sea level, will relieve the condition at once.

Treatment.—Remedial measures are simple. There can be no doubt but that warm clothing giving a sense of comfort and relief from the disagreeable chill is an important adjunct in treatment. A saline laxative should be administered and the patient placed on a milk diet, as in sprue, until the symptoms have ameliorated.

Low Country Morning Diarrhea

This is an affection not uncommonly experienced by persons on arriving in the tropics. It has been described by Castellani as occurring in Ceylon, but it is observed in other tropical locations at sea level. The day after arrival and for several days afterwards the patient will be annoyed by having several evacuations early in the morning before or after breakfast and at times before arising. The desire to go to stool is very urgent, but defecation is without pain. The stools are fluid and bile stained. The condition usually clears up in a few days but may persist for weeks in some persons. Removal to regions in which the climate in respect to rainfall, etc., is different will cause the affection to disappear.

VERRUGA PERUVIANA

RICHARD P. STRONG

Prophylaxis, and Nature of the Disease and Distribution.—Verruga peruviana is a disease particularly characterized by an eruption, which develops on the skin and occasionally on the mucous membranes, especially of the mouth and throat. At the onset there is usually malaise, lassitude, depression, pains in the joints, and fever. These symptoms are followed by the cutaneous lesions, which vary greatly in appearance. The distribution of the cutaneous eruption resembles somewhat that seen in yaws; but in many other respects the lesions of the disease are entirely distinct. In uncomplicated cases, neither the parasites of Oroya fever nor those of malaria are present in the blood; although as verruga peruviana is contracted in regions in which Oroya fever and malaria are common diseases among the inhabitants, and where visitors are likely to contract such maladies, it is not surprising that concomitant infections with these parasites frequently occur. Indeed, according to the statistics of Peruvian physicians, in a large percentage of verruga peruviana cases the blood examination shows an infection with malarial parasites; and in 1 of the 20 cases of verruga peruviana which the author studied a concomitant infection with Oroya fever was observed.

The various cutaneous lesions of the disease will be described very briefly. The eruption consists at first of erythematous areas in which groups of small papules soon form. Often small vesicles appear which may be grayish or reddish, or dark blue, according to their degree of vascularity and the character of the blood within them. The areas over which this so-called miliary rash appears are frequently edematous, and this is most commonly observed on the legs. Unless the disease is arrested the papules grow until they usually measure from 1 to about 5 mm. in diameter. They are usually reddened, raised, and sharply circumscribed from the surrounding tissue, usually discrete, sometimes confluent, and, during the most active process of the disease, are of a bright or dark cherry color. The skin over them is tense, translucent and adherent. Later they assume a gray color or become the color of the skin. As the disease advances, in addition to the cutaneous papules, deeper subcutaneous nodules appear, which at first may be quite free from the skin and lie deep in the subcutaneous tissue. Later many of these grow toward the surface and the skin over them becomes involved and adherent. These nodules vary from several millimeters to 3 or 4 centimeters in diameter. The nodular type is especially common about the flexures of the knees and elbows and over the thighs and legs. After these nodules have become adherent to the skin, the skin over them sometimes becomes broken and they may ulcerate, reaching the surface as red fungating masses. These lesions are known as verrugas of the "mulaire" type and often measure several centimeters in diameter. They also may be pedunculated. While the eruption occurs most commonly on the legs and arms and shoulders, it not infrequently appears on the face, forehead and ears. Sometimes it appears on the trunk. The miliary eruption also may occur in the mucous membrane of the mouth. After lasting for several weeks and sometimes for three or four months or even longer, during which time the eruption and nodules may disappear and reappear (that is, true relapses occur), the eruption fades, dries up, and the nodules become absorbed without leaving a scar. In some cases only the miliary eruption appears and convalescence supervenes after several weeks.

The malady has been stated to be confined to the western slopes of the Andes in Peru, though it probably occurs occasionally in southern Ecuador and northern Chili. In a case described by Haviland Hall the disease was contracted in Zaruma, Ecuador. It also seems very probable it is related to, if not identical with, the disease "angiofibroma cutis circumscriptum contagiosum" described by Bassewitz as occurring in southern Brazil. In Peru the disease is usually confined to the deep-cleft narrow valleys of the Andes lying between 2,000 to 9,000 feet above the level of the sea.

The unusual climatic conditions which prevail in these localities—situated as they are so near to the equator and isolated by mountain ranges—have undoubtedly exerted an influence in producing in some respects a very unusual regional flora and fauna; therefore it is not surprising, perhaps, that unusual diseases should be encountered in these localities. The days are very hot and sunny, but the sun sets behind the mountains at three or four o'clock in the afternoon and the nights are usually cold. There is frequently a diurnal variation of from 25° to 30° F. Cold mountain streams run through these valleys and the vegetation is abundant only in the vicinity of their banks, owing to the fact that very little rain falls in these regions. The mountain sides themselves are formed of bare rock and are almost barren of vegetable growth. The disease in the endemic regions occurs particularly among the poor who live under very unfavorable hygienic conditions and are often impaired in health and weakened by other diseases.

Age, Sex, and Race.—Apparently age, sex, and race have no influence upon the occurrence of verruga peruviana. It is often seen in newborn infants. Nearly every one who lives in the infected areas acquires the disease, and one attack usually confers immunity. In some cases in which reinfection occurs, the disease then appears in a mild form. Exacerbations or relapses, however, during the course of the infection are not uncommon. Strangers remaining for any length of time in the endemic regions in close association with the inhabitants usually become infected.

Etiology.—So far no definite visible microörganism has been discovered in verruga peruviana, although Mayer, Rocha-Lima, and Werner in the study of a single case reported the presence of bodies resembling Chlamydozoa in the lesions; and Rocha-Lima in his most recent work upon the subject includes verruga peruviana among the diseases caused by a Chlamydozoa. These observations, however, were not confirmed either by the Harvard Commission, who studied the disease in Peru, or by several other investigators. Schilling-Torgau places both verruga peruviana and variola in the list of diseases caused by filtrable viruses, but he gives no evidence to support the view that the virus of verruga is filtrable. Neither Mayer, Rocha-Lima and Werner, nor the Harvard Commission, in a few experiments, could demonstrate that the virus of verruga was filtrable.

If the skin over the eye of a monkey is scarified and a small portion of a verruga nodule rubbed into the abrasions, after an incubative period usually of from ten to twenty days, an eruption consisting of small papules appears. These gradually enlarge and later assume the appearance of the verruga nodules observed in human cases of infection. The histological structure of such lesions is similar to that of the nodule observed in man. The infection may be transmitted from monkey to monkey through a long series of animals. However, when a monkey is inoculated with the virus, no generalized eruption occurs, and, just as is the case when the animal is inoculated with small-pox virus, only a modified form of the infection develops.

The method of transmission in nature is not as yet known. It seems possible that some arthropod may be concerned, or that the disease may be transmitted in a similar manner to small-pox. However, the disease is not contagious in the sense that small-pox is. A modified form of the infection may be transmitted by direct inoculation, not only to monkeys but also to man.

Prophylaxis.—Much is obscure with reference to prophylaxis since we do not know definitely how the disease is conveyed. As the infection is usually if not exclusively contracted in the endemic areas, these should be avoided as far as practicable. Odriozola particularly emphasizes the danger of strangers remaining for a long period of time in the regions where verruga exists and that they should particularly avoid regions where turning of the soil, as in agricultural pursuits, is taking place. He also thinks that much could probably be done in the prevention of the disease in the endemic centers by drainage. Until we know more about the transmission of the infection, any abrasions of the skin or insect bites contracted in such areas should be immediately painted with tincture of iodin. Monkeys may be successfully immunized by a single cutaneous inoculation of verruga virus, and when such monkeys are reinoculated no lesions develop. Since it is the general opinion that one attack of verruga peruviana confers immunity, it has been suggested that, by using a virus after several passages through monkeys, man might be successfully vaccinated against this disease in a similar manner to that employed against small-pox. However, no human experiments in vaccination have apparently as yet been performed.

Treatment.—The natives in the endemic regions have recommended many local herbs for treatment, but none of these has been demonstrated to have a specific or definite favorable effect on the disease or its course. A substance which has a great reputation locally among the people as a specific is Buttneria cordata (family Buttneriaceæ). This plant has stimulating and diaphoretic properties and is said to act as a mild tonic. Its specific action in verruga is very questionable. Antunez has particularly recommended for treatment plants known locally as "quisuar" (Budleja incana; family Scrophulariaceæ) and "molle" (Schinus molle). Odriozola, however, has not found it to be of particular benefit, although it is also a very popular remedy in Peru. Quinin, antipyrin, salicylic acid, sodium salicylate, iodid of potassium, iodoform, salol, carbolic acid, ergotin, ammonia, mercury, belladonna, benzoate of soda, adrenalin, and thyroid extract have all been employed extensively in the treatment of verruga, but apparently without beneficial results. Arsphenamin is also apparently without effect in curing the affection. Arce treated 5 cases only 2 of which were cured. The treatment must be largely symptomatic. It should be particularly directed toward the improvement of the general condition of the patient. It is advisable to remove him from the endemic district in which he has contracted the disease. Complications should be sought for in all cases, particularly where fever is present, and treated accordingly. Malaria or Oroya fever may be concomitant infections. With reference to the treatment of the eruption upon the skin, the Peruvian physicians generally believe that cold baths may have an unfavorable effect and may cause fever and pain in the joints. Suppression of the eruption may occur, and the disease may result fatally. Martinez, however, particularly recommends treatment with thermo-baths, and claims good results from those which have radio-active properties. He has also tried the effect of autohemotherapy in the treatment of the disease without beneficial effect. Odriozola believes that, if the miliary eruption is general, no treatment is necessary as the course of such cases is usually favorable. In the cases with advanced mulaire lesions, these should be cleansed daily with a mild antiseptic solution and dressed with boric acid, zinc oxid, or some similar ointment. If the lesions have extensively ulcerated and become gangrenous, hemorrhages of considerable size may sometimes occur. In order to avoid such a complication, extirpation of the lesions may sometimes be performed with advantage.

OROYA FEVER

Richard P. Strong

Prophylaxis.—Oroya fever may be defined as a specific febrile infection, irregular in course, and usually characterized by a rapid pernicious anemia resulting in extreme prostration and frequently in death. In severe infections the patient may not live longer than from three to four weeks from the onset of symptoms.

Age and sex appear to have no influence upon the incidence of the infection. The incubative period varies between two and three weeks, the disease being introduced by malaise, pain and weakness in the limbs, and indisposition. Following these symptoms slight rigors and fever appear, which usually increase in severity as the disease progresses. The fever is very irregular; the temperature often fluctuates between 100° and 102° F., rarely rising to 104°. The fever is accompanied by a rapid pernicious anemia which may be complicated by hemorrhage, vertigo, or syncope. The skin becomes pale, the mucous membranes assume a waxy appearance, and murmurs are heard over the heart and cervical vessels. Edema of the legs and about the joints may occur. The urine is scanty and the specific gravity high, and albumin is occasionally present. The prostration increases as the disease progresses, and restlessness, insomnia, and delirium may develop. The spleen is frequently enlarged but not always palpable. The lymphatic glands are almost always more or less enlarged.

The most striking and characteristic features of the disease are found in the blood. Sometimes the red blood-corpuscles number less than 1,000,000 per cubic millimeter, and the entire course of the disease terminating in death may be less than a month. Cases have been reported in which the number of red cells fell to 500,000 per cubic millimeter in a very short period of time, death following a few days later. Nucleated red cells appear in the peripheral circulation early, and rapidly increase in number. Normoblasts are most numerous, but megaloblasts are frequently seen. Polychromatophilia and poikilocytosis are particularly marked in severe infections in both erythrocytes and erythroblasts. Many granular cells are visible and great variation in the size of the erythrocytes also occurs. A leukocytosis is almost always present, a count of 20,000 being common. The differential leukocyte count shows nothing characteristic and is practically normal. The hemoglobin varies between 15 per cent in very severe cases to 50 per cent in less severe ones.

In severe cases the parasite Bartonella bacilliformis which gives rise to the disease is present in great numbers in the red blood-corpuscles, but in mild ones a long search is often necessary to disclose the organisms. In fresh blood specimens the parasites are frequently difficult to detect. They may, however, show definite motility within the cell. In shape they are round or rod-shaped, though the rods are not always straight in outline. In preparations stained with Giemsa's solution the rod-shaped forms measure approximately from 1 to 2μ in length, and from 0.2 to 0.5μ in thickness. They are frequently slightly curved and occur singly or end to end in pairs or long chains of three, four, and five. In the endothelial cells of the enlarged lymphatic glands and spleen these same bacillary bodies are also observed. Sometimes these cells are distended also with rounded masses or spheres quite uniform in size, each of which contains a definite number of minute points of chromatin. Apparently these points later become elongated and finally appear as distinct rods. The rod-shaped elements which have escaped from ruptured endothelial cells apparently later enter the red blood-corpuscles. The parasite causing the disease owing to its resemblance to Lymphohaemocytozoön parvum has been classified as a protozoön. Hyashi has recently described somewhat similar organisms in the lymphocytes and endothelial phagocytes of the spleen and lymph-nodes of patients suffering with tsutsugamushi disease. So far the attempts both to infect animals with the blood of Oroya fever cases or the cultivation of the parasite from the blood have been unsuccessful. The transmission of the disease has also not been satisfactorily demonstrated. It seems not unlikely that the infection is carried by some blood-sucking arthropod. Townsend, who has studied the disease in Peru, believes that Phlebotomus transmits it, but other investigators incline to the belief that a mosquito carries the infection. The local physicians particularly emphasize the danger of infection from spending a night in endemic districts. If the district is left before sunset this danger is said to be very much less.

At the present time our knowledge of the geographical distribution of Oroya fever is probably very incomplete. It occurs particularly in many of the deep cleft narrow valleys situated on the western slope of the Peruvian Andes at altitudes of from 2,500 to 8,000 feet. Like malaria it appears undoubtedly to have a seasonal prevalence. It is much more prevalent from January to April in Peru, and particularly towards the close of the warm rainy season. The climatic conditions in the valleys in which the disease is endemic are peculiar and different from those in other parts of Peru. The hygienic conditions under which the majority of the people live in these districts are usually very poor, and frequently no provision is made at night for protection against insect bites.

Personal Prophylaxis and Treatment.—Provisions for prophylaxis are obscure since the exact method of transmission of the disease is unknown. Nevertheless, travelers should avoid, if possible, remaining in the endemic districts particularly at night, and, if compelled to remain, should be indoors before sunset and protected from biting insects by mosquito netting or screening.

No specific treatment for Oroya fever is known. Patients should be removed from the endemic areas unless already very ill, and placed under the best hygienic conditions and protected against cold when in the mountain regions. The disease must be treated symptomatically. All cases should be confined to bed and given light nourishing diet. The diet should be as liberal as possible in the individual case and should include a generous proportion of meat proteids, and the patient should be encouraged to take large amounts of fluids. Mild cases with moderate anemia require little treatment beyond that destined to maintain the general strength of the patient, and the administration of some iron preparation and a bitter tonic which may be given by the mouth. Fowler's solution, 2 to 3 drops three times a day, and increased until slight symptoms occur, would appear to be sometimes beneficial. If the case is of a more severe nature with fever and marked anemia, no medicine should be given orally, but iron should be administered hypodermically in the form of the green citrate, in doses of 1½ gr. (0.1 gm.) given daily for a few days and then every other day. In the very grave cases transfusion should always be considered. Moderate amounts of blood, 500 c.c., may be injected not only on a single occasion but on several, at intervals suggested by the condition of the individual patient. Whenever the hemoglobin reaches as low as from 30 to 40 per cent, transfusion is usually indicated. Odriozola has recommended in the severe cases injections hypodermically of Hayem's serum and arsenic in addition to iron. In cases where a suitable donor is not at hand for blood transfusion, intravenous injections of saline solution containing gum acacia may be employed. For the more severe cases arsenic may also be administered subcutaneously in the form of sodium cacodylate, 0.5 to 2 gr. (0.03 to 0.12 gm.) being given three or four times a week. Arce has suggested three or four doses of neo-arsphenamin given intravenously over a period of four or five days. Complications and concurrent infections should be sought for and carefully treated. Infection with ankylostoma and malaria, which may also contribute to the anemia, are common diseases in the endemic Oroya fever regions.

TROPICAL LOCAL INFLAMMATIONS

Samuel T. Darling

Ulcerating Granuloma of the Pudenda.—This affection, called also granuloma inguinale, is undoubtedly a sexual disease and attacks both sexes after puberty, causing what might better be called pudendal granuloma. Hoffman believes that it begins with blisters on the sexual organs. The glans penis, foreskin and vulva become involved and the lesion may spread to the groin, scrotum, perineum and anus.

The blister, and later, an indurated nodule, breaks down and ulcerates and a spreading granulated surface, with, in females, long irregular digitations of the labia occur. Usually confined to the pudic area, it has been described as occurring on the face and neck. The disease is widespread in the tropics yet the incidence is not high. According to Reed and Wolf, the disease has existed for many years among negroes in the Southern States.

Two types of organisms have been described as causing the lesions: (1) Donovan's bodies or Calimmato-bacterium-granulomatis. These are encapsulated bacterial bodies found within the proliferated connective tissue cells of the granuloma. (2) Wise and Cleland believe that a spirochete, S. aboriginalis, is the cause. Spirochetes, however, are extraordinarily abundant on most superficial ulcerative lesions in the tropics.

Treatment.—A number of observers have reported satisfactory results following the use of tartar emetic. The drug was administered intramuscularly to cases in which Donovan's corpuscles were found in the ulcers.

℞	Tartar emetic	gr. viij
	Acid. carbol.	minims x
	Glycerin	drams iii
	Aq. dest. ad	oz. i

Nine minims are injected into the buttocks at intervals of two days. Intravenous injections of 1:1,000 solution of tartar emetic have given good results in from eight to fifteen inoculations (see precautions for using tartar emetic under Leishmaniosis).

At the same time tartar emetic was given by mouth, the following mixture being used:

℞	Tartar emetic	gr. v
	Sod. bicarb.	gr. xxx
	Glycerin	oz. i
	Aq. chloroformi	oz. ij
	Aq.	oz. iij

Healing was soon evident and complete in six weeks.

The X-ray is said to be effective in early cases. Five or six applications of 5 H-units at intervals of three days are advised. The digitations should be excised from the vulva. Mercury and iodids are of no value in this affection.

Tropical Ulcer.—This is a sloughing ulcer which on occasion may in undernourished and severely debilitated persons become phagedenic in character. It is found everywhere in the tropics.

The affection is most common among the poorer classes of the population in the native villages at times or under circumstances of famine, where most of the population are underfed and go about with their ulcerated limbs exposed.

Vincent, Prowazek and others have described spirochetes and fusiform bacilli in the lesions, and S. vincenti is regarded as the cause by Wolbach, for these forms are found in the deeper layers of the ulcer. Important as spirochetes may be in the causation of the peculiar histopathology, there are important predisposing causes, the most active of these being undernourishment and anemia due to hookworm infection. It is the starving villager, after a famine or long dry season, and the vagabond who present the worst examples of this affection.

Indeed the cases of extensive phagedena involving large areas of muscle tendons even to the periosteum, occur more frequently among the vagabond class than any other.

The ulcer begins as a small yellow blister usually in the lower third of the leg and always below the waist. There is a widening red areola, which soon breaks down, increasing in size and becoming circumscribed as a circular area with infiltration of the underlying tissues. Necrosis occurs and a sloughing mass is gradually thrown off—leaving necrotic margins which slowly granulate. Ulceration occurs over muscles and never over bony processes. In untreated and uncared for cases a condition resembling the descriptions of "hospital gangrene" may spread rapidly from the primary area of necrosis. The ulcer, if neglected, has a tendency to enlarge and remain for months and years.

Treatment.—In backward communities, where many of these cases exist, the spread of the affection has been arrested by merely covering with protective dressings the ulcers of all the affected persons in the village. It has appeared as though the virus of the disease were spread in these villages by flies transmitting the virus from ulcers to fresh cuts on other anemic and debilitated persons.

It is of fundamental importance that the physical condition of these patients be improved without delay. Rest in bed or with the leg elevated is obligatory and the patient should be placed upon the most nourishing diet. Hookworm infection, usually present, should be treated at once. The anemia is combated with iron and arsenic.

The ulcer should be protected with a wet gauze dressing containing some substance distasteful to flies, such as cresol. Continuous wet bichlorid dressings, 1 : 5,000 to 1 : 10,000, are of value to clean the surface of the ulcer. After the ulcer is clean it may be strapped every other day with adhesive strips. At each dressing the surface is cleansed with boric acid solution. At the same time tincture of iodin is applied to the floor and margins of the ulcer. The larger ulcers will require skin-grafting.

Ointments are preferred by some surgeons. Castellani recommends an ointment of protargol, 5 to 20 per cent, to be spread over the ulcer on gauze after dressing daily with bichlorid lotion.

On the theory that the disease is caused by spirochetes, arsphenamin and other organic arsenic preparations have been administered and these drugs have probably been most effective in those cases really due to syphilis, for it must be recorded that nearly one-half the leg ulcers in the tropics are caused by syphilis.

Climatic Buboes.—This is an affection of the inguinal lymph-nodes common in the tropics. The diagnosis is made by exclusion of enlargement due to other causes such as filariasis, to venereal bubo associated with venereal sore and Pestis minor or the slight involvement of the femoral nodes seen in some light cases of plague preceeding an epidemic.

In warm climates most of the inhabitants suffer from general glandular enlargement. This is probably due to frequent skin infections, particularly of the legs, from which they suffer, and is certainly not always due to syphilis or yaws. In countries where plague has never been endemic, these climatic buboes will be encountered. Cultures from the excised nodes are sterile, and sections and smears present no morphological element which can be identified with any known microörganism. Apparently the bubo follows some infection of the leg or genitalia which presents no conspicuous external lesion. Men are usually the sufferers. There is some pain, swelling and fever.

Treatment.—Complete rest and local applications of heat relieve pain. Ichthyol and belladonna ointment or applications of lead lotion to the affected parts are recommended by Castellani and Chalmers. Time is usually saved in these cases by surgical interference and excision of the enlarged nodes. Plague should be excluded before operation by aspiration of the nodes with a small syringe first moistened with a solution of sodium citrate to dilute the gland juices and prevent clotting within the syringe.

REFERENCES

UNDULANT FEVER

SAMUEL T. DARLING

Bassett-Smith, P. W. Journ. Hyg., 7, 115, 1907.
——— South African Med. Rec., No. 10, xviii, 1920.
——— Journ. Trop. Med. & Hyg., Nos. 5, 13, xxiv, 1921.
Bruce, D. Osler's System of Modern Medicine, 1907.

Cottes, Durian de. Rev. españ. de méd. cirurg., No. 15, ii, Barcelona, 1919.
Craig, C. F. Internat. Clin., iv, 89-115, 1905.
Eyre, W. H. Lancet, 88, 182, 1912.
Fleischner, Vecki, Shaw, and Meyer. Journ. Infect. Dis., No. 6, xxix, 1921.
Gentry, E. R., and Ferenbaugh, T. L. Journ. Am. Med. Ass., 57, 889, 1045, 1127, 1911.
Nicolle and Conseil. Compt. rend. Acad. d. sc., No. 17, clxxi, 1920.
Souleyre, C. Bull. et mém. Soc. méd. d. hôp. de Paris.
Ziemann. Deutsche med. Wchnschr., No. 18, xlvii, 1921.

DENGUE

PAUL C. WOOLLEY

REVISED BY SAMUEL T. DARLING

Couvy. Bull. Soc. path. exot., No. 4, xiv.

VERRUGA PERUVIANA AND OROYA FEVER

RICHARD P. STRONG

Arce. Ann. Fac. de Méd., Lima, vii, 5, 1921; i, 24, 1918.
Barton. Crón. méd., Lima, xxvi, 7, 1909.
Bassett-Smith. Trop. Dis. Bull., xix, 817, 1922.
Biffi. Arch. f. Schiffs-u. Tropen-Hyg., xii, 1, 1908.
——— Crón. méd., Lima, xx, 149, 1903.
Cole. Arch. Int. Med., x, 668, 1912.
Darling. Journ. Am. Med. Ass., lvii, 2071, 1911.
Galli-Valerio. Centralbl. f. Bakteriol., Part I, lviii, 1911.
Gastiaburu and Rebagliati. Crón. méd., Lima, xxvi, 377, 1909.
Hayashi. Journ. Parasitol., vii, 53, 1920.
Hercelles. Ann. Fac. de méd., Lima, i, 10, 1918.
Jadassohn and Seiffert. Ztschr. f. Hyg. u. Infect., lxvi, 247, 1910.
Martinez. Apostillas, Huarez, Aug., 1922.
Mayer, Rocha-Lima, and Werner. München. med. Wchnschr., lx, 739, 1913.
Odriozola. La Maladie de Carrion ou la Verruga Péruvienne, Paris, 1898.
Rebagliati. Crón. méd., Lima, xxxii, 36, 1915.
Rocha-Lima. Mense's Handbuch der Tropenkrankheiten, 2d ed., iii, 597, Leipzig, 1914.
——— Prowazek's Handbuch der Pathogenen Protozoen, 973, Leipzig, 1920.

Strong. Nelson's Loose-Leaf Living Medicine, ii, 349D, 1921.
Strong, Tyzzer, et al. Journ. Am. Med. Ass., lxxi, 1713, 1913.
Townsend. Ibid., 1717, 1913.
——— Journ. Parasitol., ii, 143, 1916.
——— Entomol., vii, 357, 1914.
——— Science, New Series, xxxix, 99, 1914.
——— Bull. Entomol. Research, vi, 409, 1915-1916.

CHAPTER XLII

TROPICAL DISEASES DUE TO PROTOZOA

Samuel T. Darling, Hideyo Noguchi, Creighton Wellman,
William B. Wherry, and Paul G. Woolley

THE SPIROCHETOSES

THE RELAPSING FEVERS

Samuel T. Darling

While the specific treatment of relapsing fever is the same in each of the localities where various strains of spirochetes cause the disease, it is desirable to divide the clinical and other forms of the infection according to the special mode of transmission; for special means must be used in the prophylaxis of the disease, according to whether the disease is spread by lice or ticks. Two[1] types of the disease may be encountered in the United States: one a tropical variety endemic in Panama and Columbia and adjoining regions, clinically distinct, and another type usually seen in emigrants from Russia. These may be differentiated by the practitioner and should be distinguished one from the other.

The group of fevers spread by the louse are those found in northern Europe, northern Africa, India and Manchuria. The febrile reaction in this group is often distinctive and there are more spirochetes in the peripheral blood in this type than in the tropical forms. The disease can usually be diagnosed with ease from blood films and with certainty by the temperature charts.

The fevers endemic in Central and South America, in tropical Africa and in Miana, Persia, are spread through the bite of ticks, Ornithodorus talaje and moubata and A. persicus respectively.

The infection is caused by a spirochete, S. recurrentis, of which there are a number of strains, some of which have received specific names, such as S. novyi found in the cases in Panama, S. carteri in the cases from India and S. duttoni from tropical Africa. The spirochete zoölogically is placed between the bacteria and protozoa. It appears in the peripheral blood during the access of fever remaining there several days. With the crisis of temperature it disappears, having for the most part been phagocyted by endothelial cells in the liver and possibly the spleen. After an interval of several days there is a recurrence of fever and the spirochete reappears in the blood in increasing numbers until another crisis occurs. In mild forms or types of the fever there are but two or three febrile paroxysms. In more severe types, as in the African type, there may be six or more recurrences of fever and of spirochetes in the peripheral blood. In the European type there are usually only two paroxysms.

---

[1] Cases of relapsing fever have been reported from Colorado and California; they would appear to be of the type seen in tropical America.

The fever in most cases cannot be distinguished from malaria, and it may be mistaken for grip, dengue, early typhoid, yellow fever, typhus or plague. The character of the relapse should arouse suspicion, and the blood examination will reveal the spirochetes if taken during an access of fever. Animals, however, may be infected with blood taken for diagnostic purposes during the interval between paroxysms. Spirochetes are numerous in the European type and sparse in the tropical forms.

Patients are taken with severe headache, pains in the back and epigastrium and cramps in the legs; vomiting occurs. There are chills, fever and sweating as in malaria. After the fall of fever the patient feels weak. Each paroxysm repeats the features of the first, the spleen becoming enlarged and tender. The erythrocytes show basophilic change and stippling. Subnormal fluctuations of the temperature are a well-marked characteristic of this infection.

Under certain circumstances, as in times of famine and when under conditions of overcrowding, the resistance of large groups is reduced and severe types of the disease are seen. Jaundice and hemorrhages occur. Severe types exactly like typhus are seen.

Prophylaxis.—In spite of the fact that louse-borne diseases such as typhus and relapsing fever have been introduced into the United States many times, the diseases have never gained a foothold and established themselves. This is undoubtedly due to the better facilities for personal cleanliness and the better personal hygiene prevailing in this country, than in the areas from whence came the infection.

In the louse-borne types of the disease from Europe, Asia, and Northern Africa, prophylactic measures depend on the destruction of lice harbored on the person and on the clothes. The body of the infected person is cleansed with kerosene or gasoline spray, or by means of cresol soap. If the bodily hair cannot be shaved or cut short it must be thoroughly soaked with 2½ per cent carbolic acid solution. Clothing is sterilized by steaming or by boiling. Nurses and others may use underclothing impregnated with such insecticides as cresol, crude carbolic acid or naphthalene.

The relapsing fever of tropical Africa and of America is spread by ticks which are found in the native houses, huts and shelters. Europeans should never sleep in native houses, camps or any situation where natives are in the habit of sleeping. In Panama the tick Ornithodorus talaje spends the nymphal stage on the rat. Consequently in the attack on this disease in endemic regions it is necessary to eliminate the rat as well as the tick. The tick lives in the bamboo bed slats and in cracks and crevices in the houses. These must receive attention. Beds and houses should be so constructed that there will be a minimum of places in which the ticks can hide. Hammocks afford some protection but ticks, like bedbugs, will fall from the roof on to the sleeper. All travelers from endemic regions should be detained for disinfection of persons and baggage.

Treatment and Medication.—The disease is easily controlled by arsphenamin or its substitutes, neo-arsphenamin, arsenobenzol or salvarsan.

In the Persian type with sustained fever during a paroxysm, arsenobillion 0.6 gm. given intramuscularly will cause the fever to fall in from six to ninety-six hours. When from 0.3 to 0.6 gm. are given, spirochetes will disappear from the peripheral blood as early as four hours after the exhibition of the drug and the temperature will drop three or four hours afterwards. The arsenical drug should be given during the first paroxysm if possible. When too small a dose has been given a relapse will occur.

Symptomatic Treatment.—Headache and backache may be relieved by the ice-cap, and to some extent by means of phenacetin. More severe pains in the joints demand opium and codein or aspirin. When the temperature rises above 103° F. a tepid or cold sponge bath may be given. The constipation which sometimes occurs during a paroxysm may be relieved by saline laxatives.

Abortion is very likely to occur in pregnant females and should be anticipated. Famine-stricken and other debilitated persons may suffer collapse at the fall of temperature and there may be extreme weakness during the interval between paroxysms. Strychnin and digitalis will therefore be required to prevent serious symptoms developing. The enlarged spleen must not be unduly exposed to pressure for it is likely to rupture.

Specific treatment is indicated the moment the diagnosis is made by blood examination. Levaditi, however, believes that in this disease there is an opportune moment when the best therapeutic result can be achieved. This is just before crisis and he believes that the administration of the drug at this moment will enable smaller doses to be used and with a more effective riddance of the spirochete from the blood-stream.

FRAMBESIA TROPICA

PAUL G. WOOLLEY

REVISED BY SAMUEL T. DARLING

Frambesia tropica or yaws is a specific infectious disease very similar to syphilis in its manifestations. It is, as a rule, the result of inoculation at breaks in continuity of the skin, and, like syphilis, is characterized by three stages. It differs from syphilis in that it is the result of invasion of the body by an organism (Treponema pertenue Castellani), which though extremely difficult to distinguish from Treponema pallida, is nevertheless different. It also differs from syphilis in not being in any sense hereditary or derived through sexual congress, and in that the cutaneous manifestations are extremely uniform. There is a positive Wassermann in frambesia. Harper and others in Fiji believe that cases with symptoms of tabes and general paresis occur among natives who have had yaws but not syphilis.

In past centuries it is possible that frambesia was not uncommon in temperate zones. Castellani suggests that at one time it was epidemic in Scotland and Ireland. It is also possible that this disease, or one of a very similar nature, exists at the present time in Greece. But, with this one possible exception, frambesia is a disease of tropical and subtropical localities, and, when cases are occasionally seen in other latitudes, they are imported examples. Such cases appear most frequently among soldiers and sailors who have served in infected districts.

The term "frambesia" was first used by Sauvages (1750) to indicate the resemblance of the typical lesions to the raspberry (Fr. *framboise*). Charlouis (1882), to whom we owe our first definite knowledge of the relation which exists between frambesia and syphilis, used the designation *polypapilloma tropicum*. In Brazil the term *bubas* or *bobas* is in use.

Very soon after the first description of frambesia, the suspicion was entertained that this disease bore an intimate relation to syphilis; either that it was syphilis modified by a racial immunity which was the result of years or centuries of infection, or that it was the syphilis itself modified by climatic conditions. As early as 1848 Paulet showed that inoculation was possible and that typical lesions developed in from twelve to twenty days after infection, and of this fact Charlouis made use when later he inoculated successfully with syphilis a native who was affected with frambesia, and observed the development of the lesions of both diseases side by side, and showed that the course of neither disease was modified by the other. Still later Neisser in his series of experiments with apes and monkeys corroborated the earlier work of Paulet and Charlouis, and showed that each disease gave rise to an immunity which had no effect upon the course of the other; and, furthermore, he also demonstrated, by means of the Wassermann reaction, that the antigens of frambesia and syphilis are distinct. Infection would appear to be most often the result of direct contact of the virus with the abraded skin.

The rôle of insects has been promulgated by many and investigated by a few writers. And, as Gudger says, the theory of the rôle of insects, especially of flies, is a very old one. Gudger quotes from the account of Koster's travels in Brazil (1809-1815) as follows:

> "This horrible disorder is contracted by inhabiting the same room with the patient, and by inoculation; this is effected by means of a small fly, from which every precaution is oftentimes of no avail. . . . If one of them chances to settle in the corner of the eye or mouth, or on the most trifling scratch, it is enough to inoculate with bobas, if the insect comes from a person who labors under the disease."

A still earlier account Gudger quotes from Stedman's travels (1796) as follows:

> "Indeed, if a fly, which has been feeding upon the diseased, lights upon the slightest scratch on a healthy person, it communicates this dreadful disease."

Castellani's experiments, while not at all conclusive, hint at the possibility of the intermediary part played by insects. Robertson, by careful attention to bodily cleanliness among yaws patients, and by smearing their sores with mercurial ointment, has prevented the spread of infection among hospital patients, and he suggests that this result has been due to the fact that flies could not become infected by feeding at the lesions. It seems from the collected evidence that, if insects are able to convey the virus, they do it in a mechanical way, and that they are in no sense hosts. And, when we know that the secretion of the frambesial sores contains the treponemas, it should not be remarkable if insects might transmit the organism, provided these are deposited upon open wounds.

Patients usually present themselves in the secondary stage with an eruption on the face or the body, but oftentimes a primary lesion may be seen. This is not of the nature of a chancre but is usually regarded as having been a papule or papules arising on any older sore, vaccination or other scratch, abrasion or insect bite. In countries where the body is covered, the face and neck are the usual sites of the lesion, but where the people are nearly naked, the lesion may appear on the trunk or limbs. Castellani believes that the primary lesion has distinctive characters. Usually, however, there does not appear to have been any difference between the primary lesion and the secondary ones except that the latter are seen in the stage of granulomata while the primary lesion has become ulcerated and is seen in the stage of healing.

The secondary lesions are often widely though sparsely distributed, as raspberrylike granulomatous nodules covered with a soft yellow waxy crust, beneath which oozes an almost clear serous fluid. New lesions appear and older ones heal, leaving hard, dry, pigmented circles with light-colored raised margins. A variety of lesions have been described, possibly due to different viruses and to peculiarities in the constitution of the host.

Years after the secondary lesions may have entirely disappeared the so-called tertiary lesions may appear. There are circumstances, however, in which the tertiary stages emerge from the secondary without any decided interval of time.

The lesions of the tertiary stage are gummatous and ulcerative in character, involving the deeper tissues and bone. There has been some difference of opinion as to the specific character of these tertiary lesions, but two of them respond to specific treatment and must be regarded as very late manifestations of yaws. The first of these is the solitary perforating ulcer of the foot so common in Fiji where every native acquires yaws, and the other is the disease known as gangosa.

This disease was described by Leys from the Island of Guam as rhinopharyngitis mutilans. It is characterized by an ulceration of the nose, palate and pharynx involving the adjoining skin of the nose and mouth and spreading to the larynx. According to Kerr, there are extensive ulcerations of the skin and underlying connective tissues and nodular lesions of bone. The disease is found in regions where yaws is endemic.

A positive Wassermann was obtained in 85 per cent of the cases by Crow and the thorough treatment of yaws on the Island of Guam by arsenical drugs is tending to limit the number of new cases; besides Treponema pertenue has been found in gangosa lesions.

Symptoms.—Most patients with frambesia are merely annoyed by the unsightliness of the lesions and the constant oozing of serum from beneath the crusts. There may, however, be some malaise, headache and fever, with pains in the muscles and joints. Among poorly nourished and famine-stricken peoples the symptoms and lesions are more severe.

Treatment.—This is specific not only in the earlier stages of the disease but in the tertiary lesions, particularly in cases of perforating ulcer of the foot.

Arsphenamin or any of its substitutes will give most excellent results. Cases with heavy eruption and from fifteen to twenty spirochetes to the dark ground field were healed in eight days after the administration of 0.6 gm. of arsphenamin. The lesions clear up as rapidly if not more so than those of secondary syphilis. The dose for adults required of these arsenicals will vary from 0.4 to 0.6 gm.; sometimes a second dose will be required.

Among native populations it is customary to assemble the cases of yaws on certain days at hospitals and dispensaries and give the injections. Under these circumstances the dose is divided, about 0.35 gm. being given at a time. Two or at most three injections are all that are usually necessary in the cases with ulceration. The effect of these injections on the condition of native people has been most beneficial, and they are not slow to take advantage of them. In Fiji all late manifestations of frambesia, including gangosa, are included in these treatments.

Treatment by means of arsenicals should be placed within reach of all native people as a public health measure. Where this is impossible it has been recommended by Castellani to give the following mixture by mouth:

℞	Tartar emetic	gr. i
	Potass. iodid.	dr. i
	Sodii salicyl.	gr. x
	Sodii bicarb.	gr. xv
	Aq. vel aq. chlorof. ad	oz. i

One ounce is given three times daily, diluted three or four times with water to persons over fourteen years of age; half doses to children of eight to fourteen years; and one-third or less to younger children. It is recommended to give half doses to Europeans. This is given for from ten to fifteen days, discontinued for a week and given again for another ten to fifteen days.

Local Treatment.—Ulcers may be cleansed with boric solution and covered with a moist bichlorid dressing (1:1,000).

Prophylaxis.—Wherever possible cases of yaws should be isolated in screened rooms until their lesions have been sterilized by means of specific medication. Ulcers should be covered with some protective dressing containing a substance such as cresol or kreso distasteful to flies. In endemic regions it is prudent to cover all breaks in the continuity of the skin by means of some protective dressing, so as to prevent access of flies or other unknown means of spreading infection.

YELLOW FEVER

HIDEYO NOGUCHI

Yellow fever is an acute infectious disease which is now confined to tropical America and the west coast of Africa, although it has often in the past invaded North America and Europe. The virus, a filter-passer, is contained in the blood of the patient and is carried to other persons by females of the species of mosquito known as Aedes (Stegomyia) aegypti, Linnæus. It was suspected that the causative microörganism would prove to belong to the family of spirochætæ (Schaudinn, Novy, Marchoux), and in 1907 Stimson found, in a section of the kidney of a person who died of yellow fever in the New Orleans epidemic of 1905, an organism suggestive of a spirochæta to which he gave the provisional name of Spirochæta interrogans. In 1918 Noguchi isolated a spirochæta, Leptospira icteroides, from several cases of yellow fever in Guayaquil, Ecuador, and with cultures of the organism was able to reproduce all the cardinal symptoms of yellow fever in certain animals (guinea pigs, marmosets, and puppies). The pathological changes of the liver and kidney in animals dying of experimental yellow fever closely resemble those found in organs from human yellow fever. Morphologically, the icteroides resembles the organism of Stimson and also the Leptospira icterohæmorrhagiæ of infectious jaundice, discovered by Inada and Ido in 1914. Immunologically Leptospira icteroides can be differentiated from Leptospira icterohæmorrhagiæ. In animals the icteroides produces more marked jaundice and less hemorrhage than does the icterohæmorrhagiæ. The finding of Noguchi has since been confirmed in Mexico (Mérida) and Peru (Noguchi and Kligler, 1919, 1920), in Vera Cruz (Perez Grovas, 1920; Le Blanc, 1921), and in Brazil (American-Brazilian Commission, Bahia, 1924).

Yellow fever is a rapidly progressing infection. The period of incubation is from three to six days, exceptionally as long as twelve days or as short as two days.[2] The onset is sudden, with or without chills; there is an abrupt rise of temperature accompanied by severe supra-orbital headache, pains in the loins and extremities, nausea, vomiting, and intense thirst. Superficial capillary turgescence makes the face flushed and puffy and the eyes bloodshot. Epistaxis is frequent. The tongue is coated on the dorsal surface, the tip and edge are red; the gums are congested. The temperature may reach 104° F. or more, remain high for from one to two days, then drop a few degrees only to rise and continue high for from one to two days longer. At this period nausea and vomiting become more severe, and in some instances early black vomit occurs. Acute nephritis soon shows itself by the presence of casts and albumin in the urine, which is reduced in volume, and almost simultaneously comes the first appearance of jaundice, which continues to increase from day to day. Hemorrhages from the stomach, intestines, uterus, and into subcutaneous tissues in various parts of the body are usual. When death occurs, it is usually on the fifth to sixth day and is preceded by total anuresis. Convulsions, hiccoughs, delirium, and coma are the terminal symptoms. In general features the disease strikingly resembles infectious jaundice (spirochetosis icterohæmorrhagica), although the symptoms develop somewhat more rapidly. Faget's sign (relative brachycardia) is present in both diseases, and the terminal symptoms in fatal cases are practically the same. At autopsy, however, the yellow fever liver shows more advanced fatty degeneration than the liver from infectious jaundice.

---

[2] In the older literature it was often said to vary from one to thirty days.

General and Symptomatic Treatment.—Early diagnosis of yellow fever is often difficult. Examination of the blood for malarial parasites does not necessarily decide the diagnosis, for malaria is often preëxistent and does not exclude yellow fever as the actual cause of the patient's illness. Whenever there is reason to suspect a given case as one of yellow fever, as for instance in a locality where yellow fever is known to be endemic or epidemic, it is important to bear in mind the complications which may arise from careless handling of a yellow fever patient and to treat all suspected cases accordingly; that is, the patient should be kept completely at rest, all movement being forbidden, if one is to avoid aggravating the nephritic condition which will invariably be present in yellow fever sooner or later. A saline purgative at onset (such as sodium sulphate) has been advocated by Carroll. Some authors recommend calomel followed by a saline purge. No food should be permitted for the first four or five days at least, for it only intensifies nausea and vomiting, but abundant water, made palatable by ice or fruit juice (lemon) or alkalinized with bicarbonate of soda [3] should be given very freely in order to relieve thirst and keep the kidneys flushed. Some recommend, in severe cases in which the urine is scanty and there is frequent vomiting, an intravenous infusion of physiological saline. Generally speaking, however, with absolute rest, no food, and plenty of water (what Carter has called the tripod on which treatment of yellow fever rests) the general or symptomatic treatment is satisfactory.

Specific Treatment.—The isolation of Leptospira icteroides from cases of yellow fever opened up a means of specific treatment by therapeutic serum as practiced in cerebrospinal meningitis and pneumonia. A powerful polyvalent antiserum against Leptospira icteroides was produced by Noguchi in horses, and the tests in animals gave such results as to encourage its use in human cases of yellow fever. Since 1919 it has been used in many cases in various epidemics. The first observations were made in Central America by Lyster and Pareja, who constituted a yellow fever commission under the late General Gorgas. Since that time 187 cases have been treated with the serum by the health authorities of Mexico, Central America, and Peru, the results showing that when the serum is administered within the first three days of illness the majority of the patients recover. The effect of the serum is manifest not only in reducing the mortality but also in rapid alleviation of subjective symptoms.

---

[3] Four gm. in 1,000 c.c. water. Sternberg recommended giving every half hour about 50 c.c. of a solution containing 10 gm. of sodium bicarbonate and 0.02 gm. of bichlorid of mercury in 1,000 c.c. of water.

Of 107 cases treated on or before the third day of disease, only 14 have died. On the other hand, injection of the serum after the first three days of illness does not appreciably reduce the mortality, which is 40 to 50 per cent under such circumstances as compared with 50 to 60 per cent in untreated cases.

Absolute rest, as emphasized in the section on symptomatic treatment, is as important for serum-treated patients as for others. Transportation from one place to another markedly aggravates the nephritic condition, and an early injection of serum may fail to save the patient, who succumbs to the anuresis due to injury of the kidneys. Although the serum is able to kill the leptospiras, it cannot repair the injuries done to the organs. It is evident, therefore, that if the serum treatment is to be beneficial it must be supplemented by the usual symptomatic treatment and proper nursing.

The precautions to be observed in administration of the serum in yellow fever are the same as in the case of other diseases in which an antiserum produced in horses is employed. While it is desirable to give as much serum as possible at once, it is necessary first to ascertain whether the patient has an unusual sensitiveness to horse serum by giving him an initial injection of 1 or 2 c.c. If no indications of ill effects follow within one hour, the total dose, 25 c.c., or more, may be given. The danger from anaphylaxis to horse serum should be borne in mind.

SPECIFIC PROPHYLAXIS OF YELLOW FEVER

HIDEYO NOGUCHI

Prophylaxis against yellow fever is of two kinds: (1) sanitary measures against the yellow fever mosquito, Stegomyia calopus, as introduced and successfully executed by Gorgas and his associates following the experimental confirmation by Reed, Carroll, Agramonte, and Lazear of Finlay's theory of Stegomyia transmission of yellow fever; and (2) the prophylactic inoculation of non-immune individuals with a vaccine prepared from pure cultures of Leptospira icteroides.

Isolation of the Patient and Destruction of Infected Mosquitoes.—The immediate measure to be taken in case of an outbreak of yellow fever is to prevent mosquitoes from access to the patient by screening of house and bed or by placing the patient in a yellow fever hospital provided with adequate screening. This is particularly important during the first three or four days of illness, when the infectivity of the blood is greatest. The second step is to destroy all mosquitoes present in the house where the case originated by the usual methods of fumigation (pyrethrum or sulphur have been used effectively, and tobacco fumes also kill the Stegomyia when properly applied). Persons in the same house, who may perhaps be in the incubation period of the disease, should be placed under observation for at least twelve days in a mosquito-proof house or isolation hospital. Passengers on board a steamer in which a case of yellow fever has occurred should be detained for observation for a similar period, and the vessel should be anchored off shore beyond the flying distance of the Stegomyia mosquito.

Personal Precautions.—Yellow fever may exist among the native population of a region where it is endemic in a mild, more or less modified form, without being recognized as yellow fever, but under precisely such circumstances a non-immune newcomer is likely to contract the disease in severe or fatal form; hence it is of the utmost importance to protect non-immune persons in these regions by the screening of all doors and windows and the use of mosquito nets over the beds. When one is out of doors or in an unscreened house, particularly at dusk, the ankles and back of the neck ought to be well covered. It is wise to wear leggings or puttees. The Stegomyia is a domestic mosquito and chooses human habitations as its own.

Antistegomyia Measures.—The number of Stegomyia mosquitoes must be kept as low as possible. Efforts are directed toward destroying the mosquitoes in the larval stage. In the earlier days of Stegomyia elimination petroleum was used as a larvicide by spreading it in a thin layer over the surface of the water and thus making it impossible for the larvæ to get the oxygen without which they soon perish. Havana, Panama, Rio de Janeiro, Vera Cruz, and New Orleans were freed of Stegomyias by this means. In recent years, however, the distribution of certain varieties of small fresh water fish [4] in the stagnant water containing the Stegomyia larvæ has been found to be effective and far less expensive as a larvicide; the fish multiply rapidly and devour the larvæ eagerly. Connor and Hanson reported excellent results in Ecuador and Peru during 1919, 1920, and 1921, and the method is now being applied successfully in other localities (for example, in Brazil, by White and Scannell). The screening of water tanks, and the destruction of such receptacles for water as may serve as breeding places for the larvæ are simultaneously carried out by the organized antimosquito squads.

Prophylactic Inoculation Against Yellow Fever.—While vigorous antistegomyia measures are steadily reducing the areas of endemic foci of yellow fever, there are certain regions—Mexico, Brazil, and the West Coast of Africa—where endemic cases still continue to occur, chiefly among newcomers from countries free from yellow fever. The mortality is usually 50 per cent or more, and it is in the interests of the class of persons whose duties take them into endemic areas from which the Stegomyia has not been driven out that prophylactic inoculation, similar in principle to that practiced against typhoid fever, was introduced by Noguchi and Pareja. The vaccine used for this purpose is prepared from cultures of Leptospira icteroides by killing them with heat (60° C. for fifteen minutes in a water bath). In earlier preparations the approximate number of organisms was 2,000,000 per cubic centimeter, but the concentration has since been increased a thousandfold, and a cubic centimeter of the present preparation contains about 2,000,000,000 organisms. Two doses of 2 c.c. each are given subcutaneously from four to six days apart. The protection does not develop fully until the end of the second week following the last inoculation, hence vaccinated individuals ought not to enter an infected area until the lapse of this period. When the site of inoculation is well massaged after the injection, no unpleasant general or local reactions occur.

---

[4] In Ecuador the Lebiasina bimaculata, known as "huaijas" or "chalquoque" and in Peru as "liza de agua dulce," Dormitator litifrons ("chalacos"), Acanthophacelus reticulatus ("millones"), Astyanax festæ ("chatas"), Curimatus troscheli G. and Prochilodus humeralis G. ("brejas"); in Peru Pygidium punctulatum piuræ E. ("life"); in the United States and Mexico the genus Gambusia is used for mosquito eradication. See Eigenmann, C. H., "Yellow Fever and Fishes," Proc. Am. Phil. Soc., lxi, 204, 1922.

From observations made during the last three years by Lyster, Pareja, Vaughn, Bailey, Vasconcelos, Casasús, Lynn, Hanson, and others in Guayaquil, Mexico, Central American countries, and Peru, in more than 13,000 vaccinated individuals, it is established that two injections of the vaccine protect against yellow fever, while a single injection protects in the majority of instances. Protection has been well demonstrated, in instances in which approximately one-half the population only has been vaccinated, by the fact that cases continue to occur among the non-vaccinated with the usual frequency and in striking contrast to the total absence of cases among the vaccinated [5] under otherwise identical conditions. Interesting observations bearing out this point were gathered in Tuxpam and Manzanillo (Mexico), Los Amates (Guatemala), and certain sections of northern Peru, where in 1920 and 1921 there was opportunity to study the effect of vaccination. Tragic instances are not wanting in which certain individuals who rejected vaccination happened to be the ones to contract the disease and die. Sometimes persons have contracted the disease between the time of the first and second inoculations or soon after the last inoculation because of entering an infected region before sufficient time had elapsed for the development of complete immunity.

The duration of the immunity conferred by vaccination is comparatively short, perhaps not more than six months.

---

[5] During the first ten to fifteen days following the last inoculation, that is, the period required for the development of immunity, vaccinated individuals are probably as susceptible as unvaccinated.

THE TRYPANOSOMIASES

CREIGHTON WELLMAN

REVISED BY SAMUEL T. DARLING

Human trypanosomiasis of Africa or sleeping sickness is a chronic disease caused by two species of trypanosomes, Trypanosoma gambiense and Trypanosoma rhodesiense, and spread by several species of tsetse flies of the genus *Glossina* found only in Africa and adjoining parts of Arabia.

The disease is confined to endemic and epizoötic regions in tropical Africa where it breaks out into epidemic form from time to time.

Duke expresses the opinion that all these pathogenic trypanosomes are polymorphous and represent one species varying in morphology in different vertebrate hosts.

The type of disease caused by T. gambiense is endemic in the basin of the Congo and to a less extent in the Niger valley and the smaller rivers emptying into the Atlantic between 18° N. and 12° S. latitude; also along the margins of Lakes Bangweolo, Victoria and Albert.

The other form caused by T. rhodesiense is not so widely distributed at the present time as that caused by T. gambiense and is mainly found in northern Rhodesia, Nyassaland and Portuguese East Africa.

Persons who have traveled, prospected, hunted or engaged in business in equatorial Africa occasionally become infected and after their return home may seek advice and treatment in some of the medical centers in America or Europe.

Cause.—Trypanosomes are blood flagellates with a flagellum, a larger nucleated body, the trophonucleus, and a smaller one, the kinetonucleus. The latter is placed in the posterior end and from it arises the flagellum which lies alongside the body of the trypanosome attached to the undulating membrane and is the source of the energy required in locomotion. The parasite is found in the peripheral blood, in the cerebrospinal fluid and in various organs and tissues, such as the lymph-nodes, spleen and various other locations invaded by the parasite.

The trypanosomes are polymorphous in that their morphology varies under different circumstances. T. rhodesiense is very much like T. gambiense in the blood of man, but when inoculated into the rat presents posterior nucleated forms. T. rhodesiense is the more virulent of the two species, for it can always be successfully inoculated into laboratory animals whereas T. gambiense does not destroy the inoculated animal, and the infection runs a more chronic course.

The disease is transmitted by the bite of Glossina, after a developmental process carried out within the organs of the fly during a period of fourteen days, after which the fly is infective and capable of transmitting the disease. Mechanical transmission can occur in interrupted feedings. Transmission is said to occur during coition from infected males.

Pathology.—The lesions in trypanosomiasis are chiefly blood destruction, necroses, and lymphoid hyperplasia with progressive and finally severe anemia in which autohemagglutination is a constant finding. The spleen becomes enlarged, and the lymph-nodes and bone marrow hyperplastic. Evidently they are concerned in the mechanism of defense and in blood regeneration.

Mott has shown that there is a severe perivascular lymphoid cell deposit in the nervous system, particularly in the lymphatics of the brain and cord. There is also chronic leptomeningitis with proliferation of endothelium of the capillaries of the pia-arachnoid and proliferation of glia cells. The cerebrospinal fluid is turbid and increased in amount. This leads to increased intracranial pressure and flattening of the convolutions, with atrophy of nerve cells and dendrons, particularly in the cerebrum and medulla.

Symptomatology.—The earliest manifestation of the disease is fever which is irregularly intermittent, and is elevated for several days at a time (three or four to ten days) and is followed by a period of apyrexia, in which, however, there may be subnormal oscillations indicating some disturbance in the internal organs. There is a corresponding acceleration of the pulse rate. The spleen increases moderately in size and the superficial lymph-nodes become palpable.

The skin next displays a variety of changes. Usually there is circumscribed edema, as of the eyelids, face and feet, with irregular patches of erythema on the back and abdomen. Coincidently eye lesions, such as iridocyclitis or other affections of the iris and ciliary body, appear. General weakness and nervous symptoms follow neuralgic pains and cramps. Orchitis may be present in some cases.

The patient next becomes unable to make the slightest effort and sits about in a stupor. A progressive atrophy ensues, the patient becoming mere skin and bones. Various palsies are common as well as psychomotor disturbances, delirium and melancholia. Death takes place in coma.

General Treatment.—This is really only of avail in those cases taken early in the disease. Hence the importance of early diagnosis, particularly in cases of infection by T. rhodesiense. Medicaments can prolong life and in a proportion of cases cure. Diagnosis is made by blood examination of fresh films for trypanosomes and to detect autohemagglutination. Enlarged superficial lymph-nodes may be punctured with a small glass syringe moistened with sodium citrate solution the better to obtain a specimen of the gland juice. Spinal puncture is made for the detection of trypanosomes in the centrifugated sediment of the fluid. Animals may be inoculated with the blood of suspects.

Specific Treatment by Vaccines or Serum.—This has not yet been attempted in man, although a few successful experiments have been made with animals.

After an epidemic has passed through a district a notable number of persons will be found to have escaped, but it is not known whether this is because of an acquired immunity or because of some natural resistance to the disease. Very likely the latter because man possesses natural resistance to some of the very common trypanosomes of animals.

While unfortunately in most cases of trypanosomiasis the patient is only temporarily relieved by arsenical medication, life can be prolonged and this is often all that can be hoped for. This difficulty in curing the disease seems to be due to a resistance acquired by the parasite to the effects of the particular drug used known as "drug fastness." It is also attributed to the seclusion of parasites in the recesses of the nervous system beyond the reach of drugs administered by the blood-stream.

The best results are obtained in acute cases seen early. Cases of long standing with nervous symptoms are invariably fatal. Atoxyl is a drug introduced by Thomas in 1905 and is still in use. The drug is given in 10 per cent aqueous solutions intramuscularly and subcutaneously. The drug may be given over long periods of time if there occur no restriction of vision or blindness. The former symptom, therefore, should be carefully looked for during treatment, and the drug suspended or considerably reduced in amount. An antimony compound may be substituted for atoxyl in this event. Castellani recommends giving from 2 to 3 gr. of atoxyl intramuscularly every third day for at least two years, or 0.3 gm. every fourth day with 0.1 gm. for a child of ten years, for the same period. Somewhat larger doses than these have been given at correspondingly longer intervals; thus Borden and Rhodain gave 0.5 gm. intramuscularly every fifth day. Atoxyl has been combined with antimony, for the researches of Plimmer and Thomas have shown that antimony possesses powerful trypanocidal effects. A course of antimony injections may be followed by one of atoxyl. Bassett-Smith had a case under observation that had been treated unsuccessfully with atoxyl, for parasites would not entirely disappear; under tartar emetic injections given each spring for three years the patient is now regarded as cured. The total dose of tartar emetic was ½ to 2 gr. of the drug, ten injections being given.

Simpson treated early cases as follows:

Six intravenous injections at three-day intervals, each injection being 6 cg. of antimony. After an interval of one month twelve intramuscular injections each of 30 cg. of atoxyl at three-day intervals. This entire course was repeated after an interval of one month, so that during the year the patient received altogether 10.8 gm. of atoxyl and 1.08 gm. of antimony.

Plimmer recommends the salt of sodium and antimony tartrate and that it be given by rectum, 2 gr. dissolved in 2 pints of water. Tartar emetic may be given intravenously in solutions from 1:100 to 1:1,000.

Extreme care should be exercised in giving intravenous injections of tartar emetic. None of the solution should be allowed to escape into the surrounding tissues because of the severe inflammatory reaction which ensues. The depression, dyspnea and collapse which may follow the injection of antimony salts may be counteracted by the use of caffein and camphor oil in subcutaneous injections. A number of arsenical preparations have been introduced into use as substitutes for atoxyl because of the toxic effects of the latter drug on the host; among these drugs are soamin, arsenophenylglycin, arsphenamin, neo-arsphenamin, ludyl and salyl, but while their toxicity has been reduced their efficacy as trypanocides is similarly diminished.

Tryparsamid is a substance possessing a high degree of therapeutic activity in experimental trypanosomiasis; it has been investigated in the field by Pearce. Permanent cure has been effected in animals, with five strains of pathogenic trypanosomes. Its chief advantage would seem to be that it may be administered by any method and in large doses. Pearce found that the drug had marked activity in destroying T. gambiense in cases of human trypanosomiasis in the Belgian Congo. Single doses intravenously administered of from 0.5 to 5.0 gm. produced peripheral sterilization of lymph glands and blood lasting from fifty-eight to one hundred and eleven days in an average of from six to twelve hours. The effect of the drug was generally beneficial in both early and advanced cases. Subjective symptoms were found to disappear, pulse and temperature became normal, the blood characters returned to their usual appearances seen in health, and there was a well-marked gain in weight. Visual disturbances were seen in the treatment of certain advanced cases, but these were usually transitory and not persistent on a return to the use of the drug. Doses of from 4.7 to 112 mg. per kg. of body weight were administered and at intervals of one to two weeks.

Marshall has introduced a new method of treatment, the intrathecal. Believing that some of the trypanosomes are protected from the action of drugs by their secluded position in the central nervous system, and that pathological states in the membrane prevent drugs circulating in the blood-stream from reaching the infective agents in the spinal fluid; furthermore that, while parasites in the blood-stream may be killed off, the infection persists in the central nervous system and reinfects the blood-stream again. Marshall injected neokharsivan intravenously. Three hours later blood was withdrawn into a sterile container and stood for twenty hours in a bowl of cold water, serum was removed and twenty minims of it injected into the spinal canal, after a corresponding amount of spinal fluid had been removed. Marshall believes that as far as he has gone the results of such serum treatment in over fifty cases appear to be better than any of the results hitherto obtained by repeated intravenous injections of arsphenamin, atoxyl or other preparations.

Conran has shown that trypanosomes may disappear in untreated cases for long periods; cures, therefore, should be accepted with reserve.

"Bayer 205" is a new trypanocide of exceptional power in so far as experimental infections in animals are concerned. Recently satisfactory results have been reported following its use in man. Yorke has reported a case of a patient who was extremely ill, and had been getting steadily worse after unsuccessful treatment with various preparations of arsenic and antimony. After treatment with Bayer 205, his bodily weight increased 18 pounds and his general condition "improved enormously." His blood remained free from trypanosomes and non-infective to rats for four months at the time of writing.

In spite of the efforts which have been directed towards the problem of specific therapy in trypanosomiasis it is apparent that only a small modicum of the cases can be actually cured. The supreme difficulty seems to inhere in the lack of some drug of peculiar constitution which will destroy the trypanosome without unduly injuring the host.

The phenomenon of drug-fastness would preclude the idea of relying exclusively on one medicament. Undoubtedly alternating courses of arsenicals, antimony or other compounds in graduated doses will enable the therapeutist to avoid the possible weaknesses inherent in chemoresistance and the effect of large toxic doses on the host. An advantage of combined treatment lies in the summation of effects on the trypanosome yet with a scattering of toxic effects on different viscera in the host.

A variety of dyes such as benzidin dyes—trypan-red and trypan-blue and others, afridol, parafuchsin and methylene-blue—have been employed, but are not effective in human trypanosomiasis. Mercury apparently does not add to the efficiency of arsenical preparations.

General Treatment.—The general treatment of the disease is most briefly summed up in the words of Manson, who remarks that "every effort should be made to keep the patient in good general health and thereby increase his resistance. Rest, warmth, and residence in a cool and healthy climate, the systematic use of quinin for any concurrent malarial infection, and the avoidance of fatigue and everything that might depress should be insisted on." Of course, such measures are impossible when dealing with savages, whose miserable surroundings can rarely be immediately ameliorated.

Preventive Measures.—It is of primary importance that an International Administrative Board of Control be established with plenary powers for restricting the movements of all classes of people within an endemic region. A rational and efficient prophylaxis requires the maintenance of a service for inspection and treatment of all the people, a careful searching out of infected persons and revisitation of the people for the purpose of detecting infected persons and for treating them. Nothing short of a permanent prophylactic service will suffice to achieve this end.

The prophylaxis of human trypanosomiasis may be said to depend upon the proper handling of three factors: trypanosome, tsetse fly, and man, both infected and healthy. The methods adopted must, therefore, aim to attack the fly, to protect healthy people, and to prevent infected persons from becoming a menace to their fellows. Among the measures employed, clearing of fly belts, segregation and treatment of the sick, removal of people residing in fly areas, and instruction of natives are the most important.

The following recommendations, made by me in 1905 looking toward the prevention of the spread of trypanosomiasis in a district in West Africa, are illustrative of what may be aimed at in drafting preventive regulations:

1. Native camps should be removed to points some distance from rivers and kept there by police. Of course, this, as a rule, meets with some native opposition; but half a dozen native soldiers stationed at such camping places would, as a rule, be sufficient to enforce such a regulation. The natives can be induced to carry water for cooking and drinking purposes some distance, as is proved by the location of many established camping places where the water supply is sometimes situated nearly a mile from the camps.
2. Squads of soldiers, laborers, and other controllable bodies of men should be passed through the fly belts at night.
3. Native quarters in cities, towns, villages, especially visited by native caravans from the interior or coast, should be moved as fast as possible from points on rivers to the hills farther away.
4. The government should not permit sleeping sickness cases and suspected cases of trypanosome fever brought from surrounding endemic districts or from other parts of Africa to be landed at the port or ports of the district to be guarded.
5. Care should be taken not to allow recognizable cases which already exist in the district to be transported either from the coast to the interior or, vice versa, through the fly belts.

Such regulations, or similar ones, when properly carried out are of considerable influence in controlling the spread of the disease.

Among more general measures, clearing of fly areas from all brush and tall grasses or weeds has been found invaluable. For a distance of fifty yards or more from infected streams all vegetation, excepting short grass, should be cleared, and this clearing should extend up and down stream at least a hundred yards beyond the limits of the fly belt. In some districts, where the flies are very abundant, much greater areas than this should be cleared, and Wölfel found that in some districts cleared areas of even 200 or 300 meters were not enough to keep them entirely off the road. The cultivation of lemon grass (citronella citratus) on the site of old fly belts has been recommended, as has also the planting of Eucalyptus trees, these both being obnoxious to tsetses.

Another important measure is the abandoning of all fly areas. It is recommended that European houses be moved at least half a mile from the water, and, if possible, to higher ground. Natives should not be moved to points near their old haunts. They should be taken far enough away so that they cannot easily return. Cases of trypanosomiasis especially should be promptly removed from inhabited fly belts. One infected person may infect and destroy a whole village or neighborhood. Clandestine labor recruiting in Africa complicates the problem of keeping infected natives out of healthy areas. The scheme of deportation and segregation into camps where the natives are treated and prevented from returning to infected fly belts, as tried, for instance, in Uganda, has not proved successful enough to give great hope of thus stamping out sleeping sickness.

The ignorance and superstition of the native black throughout tropical Africa are formidable obstacles to any comprehensive plan of prophylaxis, as he dreads the possibility of being sent to a camp, and, in the words of Barclay, "will go to any length to avoid either himself or his relatives being segregated."

In addition to avoidance of natives, the avoidance of close proximity to animals should be recommended in trypanosomiasis districts. Parasites of the Trypanosoma gambiense type have been found in monkeys, and other animals may likewise be "sleeping sickness reservoirs."

Trypanosomes of various types have been found in horses and cattle, buffaloes, sixteen species of antelopes, hyenas, pigs, etc. Antelopes are easily infected with T. gambiense and should at least be considered as "potential reservoirs" (Bruce).

Coming now to the discussion of essentially personal precautions, it may be said that in traveling one should avoid camping in infested areas. Camps should be set at least half a mile from the edge of a fly belt. In case it is necessary to cross streams inhabited by "fly," this should be done at night, or at least early in the morning or late in the evening. When unavoidably detained in a fly belt one should take every care to avoid being bitten. The danger is especially great when fishing.

It may be pointed out here that it is very important to be able to recognize tsetse flies and to be able to distinguish them from other flies. In the absence of such knowledge the novice may unwittingly establish his camp in a deadly area. According to Austen, our best authority, with whom all African travelers will agree, "it is probable that only those who have suffered from the attacks of tsetse can recognize them on the wing, but in the resting position their identification is easy. In this attitude they can be distinguished from all other blood-sucking diptera, especially from those belonging to the genera Stomoxys and Hæmatopotus, which are most likely to be mistaken for them, by the fact that the brownish wings lie closed flat over one another down the back, like the blades of a pair of scissors, while the proboscis (that is, the proboscis ensheathed in the palpi) projects horizontally in front of the head."

The above is true of all tsetse flies, of which there are some ten species. Glossina palpalis, the carrier of sleeping sickness, is of a deeper brown than the others, so that "persons familiar with tsetse flies recognize palpalis at once by its darker coloration."

A number of tsetsefuges have been devised by different authors. Manson suggests naphthalin and camphor. Eucalyptus oil and citronella oil will either of them keep off tsetse flies for about an hour, and are of value when unavoidably detained in a fly belt.

It is said that the upper decks of vessels are much freer from "fly" than the lower decks.

Women, especially, should wear leggins or puttees when in fly areas. The tsetse fly loves to get into dark and protected places, for example, under a petticoat, and different cases of sleeping sickness in white women give a history of being bitten on the ankles.

In addition to the more general measures against the fly which have already been discussed, there has been devised by Maldonado a device for trapping adult tsetses. This is done by placing on the backs of plantation laborers a black cloth on which is plastered bird lime made from the latex of Ficus, Loranthus, and other plants. The flies are attracted by dark colors, and, descending to bite, are caught in large enough numbers to diminish sensibly their attacks.

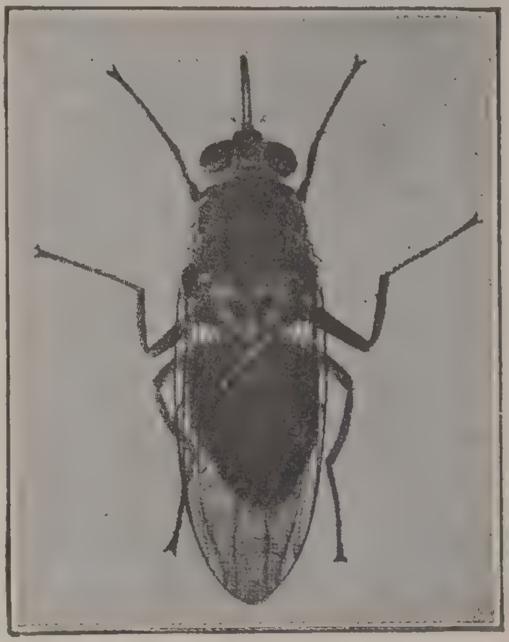


FIG. 1.—GLOSSINA PALPALIS. (After Austen.)

It must not be forgotten, while on this subject, that there are natural enemies of the tsetse, among which may be mentioned drought, birds (drongos, bee-eaters, fly-catchers, swallows, stonechats, etc.), lizards, shrews, and various insects, for example, wolf flies (Asilidæ). Possibly some natural enemy might be imported or cultivated that would eradicate the flies.

Drug Prophylaxis.—Drug prophylaxis has been studied with some attention. The treating of cases having the parasites in their blood is, of course, the most important measure. Orpiment has been recommended as a prophylactic for persons exposed, and arsenophenylglycin has been shown to be a good prophylactic in horses and cattle (Schilling). The problem of securing exposed persons against the disease by means of drug administration is a difficult one, and its solution problematical.

Clapier gave atoxyl to large numbers of natives in the French Congo who were living in infected districts. There was a reduction in mortality and apparent prolongation of the lives of many survivors of the epidemic which was a severe one.

The matter of education is an important one in the prevention of human trypanosomiasis. The prophylaxis of sleeping sickness should be carefully and simply explained to chiefs and other intelligent natives. Missionaries and others who know the native dialects can be of great service to the indigenes in this respect; for, in order to carry out the best regulations against the disease, the coöperation of the native peoples must be elicited. One of the greatest difficulties at present is their apathy and indolence. As Hodges says:

> "They seem to expect to be paid by the government for any labor which they are recommended to undertake to save themselves from extermination."

In conclusion I may sum up the things to be done in the following rules, compiled from the Sleeping Sickness Bulletins: (1) clear fly-infested areas; (2) fill up or drain pools, etc.; (3) cultivate plants noxious to fly (Citronella, Eucalyptus); (4) destroy the animals on which fly feeds (while opinion is divided as to the influence of infected wild game on the incidence of human trypanosomiasis, it would seem to be a prudent measure to remove whatever restrictions exist as to the killing of game near human habitations in fly areas); (5) encourage natural enemies of fly; (6) collect or destroy pupæ or adult flies; (7) remove villages or markets to fly-free areas; (8) discourage or forbid occupations carried on in fly belts (fishing, etc.); (9) avoid fly belts in heat of day; (10) select roads which do not traverse fly belts; (11) protect the person by suitable clothing, nets, screening, etc.; (12) use tsetsefuges when unavoidably in fly belts; (13) instruct natives in avoiding fly; (14) segregate the sick away from fly and from healthy persons; (15) prohibit moving of persons from infected to uninfected fly districts; (16) prohibit moving of infected persons from one infected district to another; (17) treat all infected persons.

HUMAN TRYPANOSOMIASIS OF SOUTH AMERICA

(Chagas' Disease)

Samuel T. Darling

This is a chronic disease caused by a blood flagellate known as Schizotrypanum cruzi which parasitizes, and is disseminated by several species of predacious biting bugs of the genus Triatoma.

The bug lives in the cracks and crevices of the walls of the houses of the people, particularly in those mud-walled hovels of the poorer classes in the endemic regions of Brazil and Venezuela.

According to Chagas who discovered the disease, transmission is effected through the bite of infected Triatoma; T. megistus and T. sordida have been found capable of disseminating the disease. Chagas alone has found Triatoma with infected salivary glands. Brumpt and Mayer could not infect animals with T. megistus by biting, although feces of the said bugs proved infectious on inoculation. Other bugs or acarids are capable of supporting the parasite, such as Ornithodorus moubata, Rhipicephalus sanguineus and Cimex lectularius.

Children are bitten and begin to develop the disease even in the first year of life. There is at first a febrile reaction rather severe in character, followed by enlargement of the lymph-nodes and spleen. The thyroid gland is said to undergo enlargement but it must be remembered that the disease occurs in a region where endemic goiter coexists. A condition of myxedema is described by Chagas as following the stage of fever. Keratitis, conjunctivitis and panophthalmia are said to characterize the disease. Myxedematous, cardiac, nervous, and suprarenal types are described according to the preponderance of clinical symptoms.

The parasite differs from trypanosomes in that multiplication takes place in the cells of the tissues and not by the usual longitudinal division in the blood-stream. Parasites are found to invade a wide variety of tissues—muscle, nerve, thyroid, suprarenal, ovary and testis.

While there is no question of the existence of human trypanosomiasis in the areas mentioned above, there seems to have been some confusion of the disease with endemic goiter, chronic malaria and possibly severe hookworm infection.

The diagnosis is made by means of blood films but oftener by animal inoculation because of the sparsity of the forms in the peripheral blood. Brumpt has introduced the method of xenodiagnosis or infection of clean Triatoma by feeding on patients suspected of having the disease.

Treatment.—If the child survives the acute stage a chronic malady lasting several years ensues. Drugs seem to be of no value in limiting the disease in animals, and treatment in human cases seems to be equally unavailing. This may be on account of some specific resistance of the parasite to such drugs as have been used, and to the inaccessibility of the reproduction forms to the medicament in the tissues of the host.

Prophylaxis.—The disease is most commonly found in the mud hovels of the very poor classes of the people in Brazil. The problem is fundamentally an educational economic one of housing at the moment, for, until the people are in a position to provide themselves with properly constructed houses which can be made vermin-free and can be maintained in this condition, little can be done. Where the disease exists in houses of better construction and possessing fewer places for the Triatoma or "Barbeiro" to live in, the people should be taught how to rid their homes of the bugs. This is being done in the schools and generally in the endemic regions to-day in Brazil. Houses with smooth entire walls free from large cracks and hiding places can be maintained free from "Barbeiro."

Escomel, in 1919, found in the blood of a person who had lived in the eastern forests of Peru a trypanosome which at first was regarded as Schiz. cruzi, but which Yorke believes is a distinct species and to which he has given the name T. escomeli.

Escomel describes the patient as having suffered for a long time from fever; the examination, however, was conducted at a time when the patient was free from fever but presented anemia and debility. There was general subcutaneous edema which appears to have been myxedematous in character in that it did not pit on pressure. The patient had experienced feelings of extreme prostration and had had attacks of somnolence.

THE LEISHMANIASES

SAMUEL T. DARLING

VISCERAL LEISHMANIASIS

Visceral leishmaniasis is a chronic disease caused by a protozoan known as Leishmania donovani, but which seems to be identical with Herpetomonas, a group of flagellates which parasitize insects.

Until recently two types of the disease were described: One is known as kala-azar or dum dum fever encountered in India, in Madras and the deltas of the Ganges and Brahmaputra; in Assam in the Garo Hills; in Central China and along the Yang-tse-Kiang River. There are endemic areas in Turkestan, Asia Minor, northern Africa (except Egypt) and in the Sudan. The other form is known as infantile kala-azar, caused by Leishmania infantum, and is found in the Mediterranean littoral, attacking infants under one year. At present it is believed that the virus of the two forms of the disease is identical although there is a preponderance of cases during infancy and early childhood in the subtropical Mediterranean and a larger attack rate among adults in the other tropical regions mentioned.

The biological cause is morphologically identical with the non-flagellated phase of certain flagellates found in the intestinal tracts of mosquitoes, flies, fleas, biting bugs and also in the latex of certain plants belonging to the Euphorbiaceæ.

In the vertebrate host the microörganism is small and spindle-shaped, 2 to 4.5 μ long by 1 to 2.5 μ wide, containing a nucleus and a smaller rod-shaped mass, the kinetonucleus or blepharoplast. The parasite multiplies by fission after invading endothelial cells of the blood and lymph vessels. In cultures the organism becomes flagellated, but in the tissues this never occurs. The widespread invasion of the endothelium of the spleen, liver, lymph-nodes and bone marrow causes hepatic and other necroses, liver cirrhosis, splenomegalia,[6] and lymphoid hyperplasia. Severe anemia and ascites are later and terminal manifestations.

There is a progressive anemia and the hemoglobin may become reduced to from 50 to 80 per cent of its normal amount. This is more profound in the infantile cases. As the tissues become invaded, an irregularly intermittent fever of septic type is induced. A cachectic state supervenes in which stomatitis, persistent diarrhea, emaciation and great physical weakness are the prominent features. Various complications occur, such as hemorrhage; petechial hemorrhages of the skin particularly are common in children. Ulceration of the intestines is due to localization of the parasite in the wall of the intestines.

Prophylaxis.—While it is true that the parasite appears in the peripheral blood, it never occurs in notable numbers; biting insects, therefore, if infected at all, would be infected with great difficulty. It is assumed that the disease is insect borne largely because of its epidemiology in Assam, and because the parasite resembles certain flagellates of insects. It is not impossible, however, that the parasite may come in the first instance from insects biting or otherwise, or from vegetable plants, or indirectly through the drinking or bathing water, and, after invading the tissues, may remain locked up within them, having no avenues or means of escape. The acquisition of the infection may therefore be prevented by any means directed against the visitation of insects likely to carry flagellates, such as mosquitoes, biting bugs, ticks, bedbugs, fleas, etc. While it may be impossible for such insects to regain the parasite from the blood or tissues of infected persons and infect others nevertheless it will be prudent to protect infected patients from the bites of these insects. Cutaneous wounds should be protected by suitable dressings containing some drug distasteful to flies, such as cresol, to keep them from voiding their droppings on the open, broken skin. Drinking water should be boiled. In other words, the virus of leishmaniasis may not be an obligate human virus and it is quite possible that it is an example of a peculiar protozoan parasite or parasites which live customarily in the intestinal tract of invertebrates or in the juices of plants or in some other medium, but which may easily adapt itself to the tissues of man giving an example of a biological blind alley. In this circumstance, while passively entering the tissues and destroying them it has no effective means for leaving the tissues and regaining its insect or vegetable environment. The patient in this case is not a source of danger to the community in that the infection cannot be transmitted from him to others, nor can the parasite leave his body by any natural means. The patient should, in the state of our ignorance regarding the means of spread of this disease, be removed from his home and isolated from presumptive source of infection. As there is some evidence of place infection native houses should be destroyed including their contained vermin. Dogs are liable to a similar disease and should be kept out of range of infected persons. In Assam, where the disease was rife in certain villages, the population was removed to contact camps in small units and kept under observation. Infected persons were treated, and presumably infected dwellings were destroyed.

---

[6] The disease must be differentiated from other forms of splenomegalia by splenic puncture: Banti's disease, splenomegalia endemic in malarial and bilharzial regions, histoplasmosis and toxoplasmosis. In the first three mentioned the splenic puncture smears are free from parasites; in histoplasmosis and toxoplasmosis the organism is blastomycotic and probably not affected by antimony medication.

Treatment.—The protozoal nature of the virus led to the use, by Vianna, of antimony in treating dermal leishmaniasis, and its use has been extended to the visceral type of leishmaniasis in the infantile as well as the Indian forms. Various forms of antimony are given and the results are generally satisfactory. The drug destroys the parasites and this brings about a reduction in size of the distended organs. As the parasites disappear, the fever declines, health is improved and the patient is ultimately cured in many cases.

Tartar emetic has been used in kala-azar by Di Cristina and Caronia. Rogers and Hume and also Knowles have used it extensively in India, although Rogers has abandoned the potassium salt for the less toxic corresponding sodium salt of antimony.

Knowles recommends the "heavy" powdered salt made up in a 1 per cent solution in normal saline and autoclaved at 110° C. for ten minutes only. Opalescent turbid solutions or those with a sediment should not be used. He begins in an adult with an intravenous injection of from 3 to 4 c.c. given on alternate days. This dosage is gradually increased until from 10 to 12 c.c. are given. In a course of treatment a total of 200 cg. are administered during a period of from two and one-half to three months.

In Jemma's Clinic in Naples, where children are the patients, a 1 per cent solution is injected intravenously, the median basilic, external jugular, or temporal veins being used. A child of three years receives as the first dose 2 c.c., increased to 3, 4, and 5 c.c. Other children receive from 2 to 3 c.c., increased to 6 and 7 c.c., giving from 30 to 50 cg. in from forty to fifty days. The drug should be given daily for a week, then every other day for a period, and finally once a week.

While there are no evidences of acquired immunity to the drug, Knowles believes that it may be taken up and retained by the liver. Toward the end of the course of treatment it is advisable, therefore, to increase the interval between doses. The solution should be warmed and very slowly injected from an all glass syringe. The 1 per cent solution is less liable to cause local irritation, thrombosis or phlebitis. Some persons suffer from cough, fever, vomiting and diarrhea and depression after taking tartar emetic. It is inadvisable to give an injection within two hours of a meal. Complications are usually due to peculiar involvement of tissue or viscera; it is advisable, therefore, to get the patient under treatment as soon as possible. Even when nephritis and dysentery are present, unless the patient is extremely ill, the drug should be cautiously administered.

Muir has reported a series in which, out of 143 cases treated, all were cured except 46 and of these 13 died and 33 discontinued treatment. He advises injections on alternate days until the fever has been absent for a month. Weekly injections were then maintained for from two to four months to avoid relapse. It has been found that a variable number of cases will recover spontaneously. Any complication that increases the polynuclear count is of favorable prognostic significance.

Vaccine treatment with cultures of the parasite have not proved of any value.

Splenectomy is too dangerous an operation in severe cases and cannot be recommended.

Rogers has introduced a sodium salt, sodium antimonyl tartrate, as being less toxic than tartar emetic but as effective in destroying the parasites. When this drug is given every third day once or twice a week, about 1 c.c. of a 2 per cent solution for every 5 kg. body weight may be used over a period of from two to three months. In delicate persons, as the aged and children, only one-third of this quantity should be given.

In treating kala-azar, cases are generally regarded as cured when the temperature has been within normal limits for from two to three months, when there has been a satisfactory gain in bodily weight, when the leukopenia has been replaced by a normal leukocyte count and the spleen has returned to nearly normal dimensions.

Hookworm disease not uncommonly causes an irregular fever like that of kala-azar, and it should be thoroughly treated before the treatment for kala-azar has been completed so as to remove this debilitating factor and prevent confusion in utilizing the temperature curve as an index of the efficacy of treatment. Malaria is a frequent cause of splenomegalia and fever, and, if present, should receive appropriate treatment.

The patient's general condition should receive attention. The blood-forming organs are frequently exhausted and daily doses of yeast internally administered have been recommended to increase the leukocytes. Tonics of iron, quinin and strychnia to improve the appetite, arsenic in the form of Fowler's solution in increasing doses and a course of freshly prepared Blaud's pills are advised. The deficient blood formation has been due in part to the presence of enormous numbers of parasites. When these have been removed the effect of rest, abundant nutritious food and fresh air alone are sufficient to enable the patient to recover ground. Among natives habituated by custom and economic reasons to a vegetable diet, convalescence is necessarily prolonged, for they often cannot procure nor utilize the foods necessary to a rapid convalescence.

Europeans should be put under treatment at once and then sent to live outside the endemic region where medication may be completed.

Uncomplicated diarrhea is often due to the localization of lesions in the intestinal tract; this clears up under medication. The diarrhea due to cachectic states is usually more intractable, but careful dieting with easily digested but nutritious food will tend to relieve the condition. When the colon is involved, irrigations of thymol in 1:2,000 solution may be used. For diarrhea suspected of being due to enteritis finely powdered thymol in gelatin capsules with sugar of milk in 0.25 to 0.5 gm. doses may be given by mouth. Large doses of bismuth are of value in altering the flora and fauna of the intestinal tract in diarrhea and dysentery. Amounts equal to 3 dr. by weight every three hours stirred into a glass of water may be taken. The greater the tendency to diarrhea the more necessary will it be to restrict the diet to milk and fluids.

Hemorrhages from the nasal passages and from the gums in adults and children require adrenalin.

The gums and buccal mucosa and occasionally the vulva are the seat of ulceration which, unless carefully treated, may develop into a fatal noma and septicemia. Antiseptic applications may be used, as well as the cautery. The condition is only found in extremely severe cases and treatment is usually unavailing.

Dermal Leishmaniasis

Synonyms.—It is known by various names in the endemic districts: Aleppo boil, Delhi sore, Clou de Gafsa, Biskra button, Oriental sore. In America it is called pian-bois (Guiana), Forest yaws BrG, Bosch yaws, Bush yaws, oreya de los chicleros (Mexico), uta (Peru), peste bouba (Brazil), espundia (Peru, America), ulcera de Baurú (Brazil).

Dermal leishmaniasis is a chronic ulcerative affection of the skin and sometimes of the nasopharyngeal mucous membranes of a specific nature and apparently caused by several varieties of protozoa similar to those found in the intestinal tracts of invertebrates and in the juices of plants belonging to the Euphorbiaceæ.

The lesions are polymorphous in character and there are undoubtedly several varieties of the virus. In the Orient, where the disease has been known for centuries in the Levant and adjoining countries, the lesion is frequently one or several papules which ulcerate, while in South America a greater variety of lesions are encountered and these are at times (20 per cent of cases in Brazil) followed by or associated with involvement of the adjoining nasal mucosa or extensive and sometimes fatal ulceration of the oropharyngeal wall with involvement of the larynx.

No sharp line can be drawn in regard to the geographical distinction of virus or type of lesions. Typical solitary "oriental sores" have been encountered sporadically in Panama, and, on the other hand, cases of involvement of the pharyngeal mucous membranes have been reported from the Orient, and the Sudan.

The affection is tropical and subtropical in distribution and has been found within the latitudes 36° N. and 23° S. and in localities having an average annual temperature of 17.6° C. or more, and in which the average winter temperature is not lower than 6.3° C. These conditions are found in several of the southern states.

Various names have been given to the protozoa encountered in the lesions: Leishmania tropica, L. brasiliensis and L. americana, but they are probably all representatives of flagellates of the genus Herpetomonas and are probably derived in the first instance from some biting or sucking insect, such as the mosquito, Phlebotomus or common fly.

The virus is a protozoan which is adapted to life in the intestinal tract of an invertebrate, or the juices of a plant, but which is capable of maintaining itself in the tissues of man. Fifteen thousand cases occurred within five years in Brazil in the area adjoining the virgin forest during the process of clearing, but the disease did not spread outside of this newly formed endemic sylvan focus.

The mode of infection is unknown but may take place by the contamination of an abrasion in the skin with the droppings of an infected insect or by the infected juice of some plant. Or it may be derived by the bite of some suctorial insect and the parasite injected at the time of biting, or by the scratching-in of the parasite deposited on the skin during the act of biting.

The parasite invades the connective tissues of the corium, and, depending on the virulence of the virus, a variety of lesions occur. Papules and nodules usually becoming ulcers are found in places where mosquitoes commonly visit, such as on the wrist and back of the hand (Stegomyia) or on the free margin of the ear (ear ulcer of the Chicleros), a lesion not uncommonly seen in Panama. The face and neck are commonly the seats of attack. Exfoliative lesions resembling ring-worm and yaws are common in South America. Extensive spreading lesions likely to be confused with lupus were encountered in Panama. The central areas healed, leaving an extensive area of cicatricial tissue which at times became keloidal in the negroid races. The parasite is found sometimes though rarely in enormous numbers, invading and distending the connective tissue and endothelial cells of the part. In the older parts of the lesion, parasites may be very sparse indeed. The duration of the affection is a matter of months and occasionally years.

Invasion of the mouth and nose and of the pharynx, ultimately involving the larynx, usually occurs after a considerable period of time. In some of the South American cases the primary dermal lesions seem to clear up entirely but to be followed a few years later by involvement of the nasal mucosa, the lesion extending backward and downward until the larynx is involved. Many cases of this affection are overlooked because of its resemblance to other specific diseases of the skin. The infection has been detected most often where the microscope is customarily used in clinical diagnosis; and it is overlooked where the use of the instrument is neglected. Smears from the margin of the ulcer, the papules, or scrapings of the lesion should be stained with Leishman-Wright-Hastings stain and examined long and carefully for the characteristic spindle-shaped bodies, which may be intracellular or lie free in the juices of the scrapings.

Prophylaxis.—We are in absolute ignorance of the mode of infection and are unable to give any advice as to a means for protection against the infection. The period of incubation is so prolonged that the recollection of the bite of an insect would have been forgotten. Still it is prudent to protect against the bite of insects in endemic localities, particularly on margins of jungle, in process of clearing, on jungle trails and in villages and cities where the disease is rife in the fly season. All superficial cuts should be covered at once with some dressing preferably containing cresol or other substance distasteful to flies. It is extremely unlikely that a fly, mosquito or other insect should be able to locate a small papule or other lesion and recover the organism and spread the disease from an infected person to some one else. This infection is very likely an example of a biological blind alley, that is, the parasite is introduced into the tissues of an unusual host and has no way of getting out and regaining its usual host. Solitary sporadic cases of the disease arise in the jungle where there are no other infected persons. The disease must therefore arise from some non-human source, and it is most reasonable for the present to assume that the natural source of reservoir is insect or vegetable and not human, and that persons are not in themselves dangerous or a source of infection to others.

Treatment.—Good results follow the excision of the nodule, papule or ulcer when it is solitary or few in number. The incision should be made beyond the area of inflammation and in healthy uninvolved skin.

The treatment is specific and was introduced by Vianna in Brazil. The drug, antimony, may be given in the form of the potassium or sodium salt and this is the only medication that can be recommended. *It should be thoroughly and conscientiously persisted in, particularly in the Amer-*

ican types of the disease, so as to avoid the very serious later involvement of the nose, pharynx and larynx.

Torres administers injections intravenously of 0.06 to 0.07 gm. as an initial dose; this is increased 0.02 gm. at a time until 0.14 gm. is reached. A week's interval is given after about fifteen injections have been given. Lesions in the mucous membranes as well as the skin are also treated locally with 0.10 per cent solution of tartar emetic. Children are given a dose in proportion to their age. Intramuscular injections may be given to children, or in cases where it is difficult to use the intravenous route, but antimony salts are very prone to cause severe pain and inflammation. Abscesses may result from their use.

Castellani recommends for intravenous use the following preparation:

Tartar emetic	gr. viij
Acid. carbolic	minims x
Glycerin.	dr. iij
Aq. dest. ad fld.	oz. i

One-half to 1 c.c. (8 to 16 minims) every other day in the gluteal region.

Hot fomentations will be found useful in relieving the pain which develops after these injections. Care should be exercised not to inject subsequently an infiltrated area.

In giving antimony salts intravenously it is important that the solutions be slightly warmed and free from turbidity or sediment. It is recommended that they be passed through the Chamberland filter or sterilized on successive days not in the autoclave. None of the solution should be permitted to escape into the surrounding tissues. Symptoms of giddiness or nausea may follow the injection (see article on Visceral Leishmaniasis).

Treatment in some cases is ineffectual and, as with visceral leishmaniasis, in these cases some other method should be tried. Some cases resistant to antimony salts are due to other causes such as error of diagnosis. Many cases slowly heal spontaneously.

From the fact that the parasite is invading the fixed connective tissue cells of the corium and of cells deep in the floor of the ulcer, ointments can probably do little specific good except to keep the superficial incrustation soft and prevent the visitation of flies to the wound. Local inunctions of antimony oxid and of tartar emetic have been recommended. Archibald advises the use of an ointment containing methylene-blue. Adams has successfully used a method of freezing for sixty seconds with carbon dioxid snow crayon.

During the War the X-ray came into use in treating sores on the face, particularly about the eye. Mitchell found that a single full pastil dose cured the majority of cases in ten days. The rays seem to reduce the tendency to necrosis and ulceration and the scars following this treatment are less conspicuous.

Photinus injects emetin hydrochlorid into and around as well as beneath the sore, using 0.10 gm. of a solution.

Evans has introduced the method of treatment by ionization or application of the galvanic current through a zinc electrode attached to the positive pole. The electrode is firmly applied to a pad half an inch thick soaked in a 2 per cent solution of zinc sulphate.

DISEASES DUE TO FLAGELLATA AND CILIATA

William B. Wherry

Balantidium Coli.—Ulcerative colitis, in which the Balantidium coli plays an important primary or secondary part, appears to be widely distributed, cases having been reported from many parts of Europe, from Asia, Africa, North and South America, and from the Philippine Islands. Strong (1904) collected and summarized the findings in 124 reported cases, including 2 of his own.

The lesions and symptoms simulate those of intestinal amebiasis. Bowman studied 10 cases in Manila. Of these 2 died. Most of the cases exhibited no symptoms excepting an occasional diarrhea. Balantidia only could be found during these attacks. He suggests that at this time "nests" of the ciliates are set free by ulceration, which perhaps follows the local plugging of blood-vessels by the parasites.

The histologic studies of Solowjew, Askanazy, Klimenko, and Strong have demonstrated beyond question the distribution of the parasites in the mucosa, submucosa, and in the blood-vessels of the submucosa. However, it is still unsettled whether the Balantidia are capable of penetrating the mucosa in the absence of a preëxisting lesion. The absence of polynuclears in the neighborhood of early lesions would seem to rule out the primary action of bacteria (Bowman). Numerous experimenters have failed to infect dogs, cats, rabbits, guinea pigs, monkeys, and pigs by the feeding or injection per rectum of infected feces.

A Balantidium, which is believed to be identical with that found in man, is found as an apparently harmless commensal in the intestinal tract of the domestic pig. Some believe that under unknown conditions man and other animals acquire their infection from the pig. That some unknown underlying conditions are necessary to prepare a host for infection or infestation may be argued from the failures to infect young healthy pigs.

Brooks (1902) concluded that an outbreak of dysentery among the orang-outangs in the New York Zoological Park was due to Balantidium coli. Noc (1908) reported natural infection in a monkey (Macacus cynomolgus). Brumpt has reported a case of Balantidium colitis in a Macacus cynomolgus. He was able to transmit the disease to six other macaci and transferred it from the monkey to the pig and from the pig to the monkey.

From a study of 111 cases reported with sufficient completeness Strong concludes that 32 (29 per cent) recovered; and that, while the mortality was apparently 30 per cent, a number of these cases died of other diseases. The same writer concludes that the "prognosis seems to depend upon the general condition of the patient, and upon whether he has sufficient strength to undergo local treatment, the extent to which the disease has progressed, and upon the coexistence or non-existence of other diseases."

Treatment.—Quinin enemata, given as directed under intestinal amebiasis, seem to have been as successful as any other method of treatment. Success seems to depend upon the patient's ability to take a sufficiently long course of treatment. Vincent has suggested the use of one-half per cent Labarraque's solution (sodium hypochlorite) given as enemas. The rapid cure of a case treated with ipecac is reported by Duncan.

REFERENCES

THE RELAPSING FEVERS

SAMUEL T. DARLING

Arkwright, Bacot, and Duncan. Tr. Soc. Trop. Med. & Hyg., 1919.

Briggs. Journ. Am. Med. Ass., lxxix, 941, 1922.

Voegtlin, Johnson, and Dyer. Pub. Health Rep., No. 45, xxxvii, 1922.

Carter, H. R. Yellow Fever, Nelson's Loose-Leaf Medicine, 1920, ii, 113.

Connor, M. E. Yellow Fever Control in Ecuador, Journ. Am. Med. Ass., lxxiv, 650, 1920; lxxv, 1184, 1920.

Eigenmann, C. H. Yellow Fever and Fishes, Proc. Am. Phil. Soc., lxi, 204, 1922.

Finlay, C. J. Trabajos selectos. República de Cuba, Secretaria de Sanidad y Beneficencia, Habana, 1912.

Gorgas, W. C. A Few General Directions with Regard to Destroying Mosquitoes, Particularly the Yellow Fever Mosquito, Senate Doc. No. 822, 1911, 61st Cong., 3d sess., 239.

——— Sanitation in Panama, D. Appleton & Co., 1915.

Hanson, H. Address before the Medical Association of the Canal Zone, Aug. 19, 1921.

Inada, R., Ido, Y., Hoki, R., Kaneko, R., and Ito, H. The Etiology, Mode of Infection, and Specific Therapy of Weil's Disease (Spirochætosis icterohæmorrhagica), Journ. Exper. Med., xxiii, 377, 1916.

Marchoux, E., Salimbeni, A., and Simond, P. L. La Fièvre Jaune, Ann. de l'Inst. Pasteur, xvii, 665, 1903.

Noguchi, H. Etiology of Yellow Fever, Journ. Exper. Med., xxix, 547, 565, 585, 1919; xxx, 1, 9, 13, 87, 95, 401, 1919; xxxi, 135, 159, 1920; xxxii, 381, 1920; xxxiii, 683, 1921; xxxvi, 357, 1922.

——— Recent Experimental Studies on Yellow Fever, Am. Journ. Hyg., i, 118, 1921.

——— Prophylaxis and Serum Therapy of Yellow Fever, Journ. Am. Med. Ass., lxxvii, 181, 1921.

——— Researches on Yellow Fever: Comparative Etiological, Pathological, and Epidemiological Considerations: Prevention and Treatment, Lancet, ccii, 1185, 1922.

Noguchi, H., and Kligler, I. J. Experimental Studies on Yellow Fever Occurring in Mérida, Yucatan, Journ. Exper. Med., xxxii, 601, 1920.

——— Immunological Studies with a Strain of Leptospira Isolated in Mérida, Yucatan, Ibid., xxxii, 627, 1920.

——— Experimental Studies on Yellow Fever in Northern Peru, Ibid., xxxiii, 239, 1921.

——— Immunology of the Peruvian Strains of Leptospira icteroides, Ibid., xxxiii, 253, 1921.

Noguchi, H., and Pareja, W. Prophylactic Inoculation against Yellow Fever, Journ. Am. Med. Ass., lxxvi, 96, 1921.

Pérez Grovas, P. Experimental Transmission of Yellow Fever, Cases Occurring in Vera Cruz in 1920, Together with the Isolation of Leptospira icteroides, Ibid., lxxvi, 362, 1921.

Reed, W., Carroll, J., Agramonte, A., and Lazear, J. W. The Etiology of Yellow Fever, Senate Doc. No. 822, 1911, 61st Cong., 3d sess., 56.

Sternberg, G. M. Report on the Etiology and Prevention of Yellow Fever, Gov't. Printing Office, 85, Washington, 1890.

Stimson, A. M. Note on an Organism Found in Yellow Fever Tissue, U. S. Pub. Health Serv. Rep., Part 1, xxii, 541, 1907.

The Trypanosomiases

Creighton Wellman

Revised by Samuel T. Darling

Brown and Pearce. Journ. Exper. Med., xxx, 437, 455.

Clapier. Bull. Soc. path. exot., xiv, 238, 1921.

Duke. Parasitology, xiii, 352, 1921.

Jacobs and Heidelberger. Journ. Exper. Med., xxx, 411.
Pearce, Louise. Ibid., No. 6, Supplement, xxxiv.
Yorke. Ann. Trop. Med. & Parasitol., No. 4, xv, 1921.

The Leishmaniases

Samuel T. Darling

Evans. Trop. Dis. Bull., xvi, 206, 1918.
Laveran, A. Leishmaniases, 521, Paris, 1907.
Sheppard, R. L. Bibliography of Leishmaniosis, Kala-Azar Bull., Sleeping Sickness Bureau, London, 1911.

CHAPTER XLIII

DISEASES DUE TO TROPICAL CESTODES, TREMATODES, AND NEMATODES

Samuel T. Darling, Creighton Wellman, and William B. Wherry

TENIASIS

Creighton Wellman

Revised by Samuel T. Darling

Infection with tropical cestodes, strictly speaking, is not a very common condition, although the cosmopolitan tapeworms are abundant enough throughout tropical and subtropical countries. The species discussed below may be considered as more or less special to hot countries.

Somatic Teniasis.—The invasion of the tissues with larval tapeworms is not uncommon in tropical and subtropical countries, and cysticercoid and hydatid diseases are more or less frequently reported, but are not deserving of special description as tropical infections. Three dibothriocephalid worms, however, should be particularly mentioned here.

1. The tapeworm, Sparganum mansoni, in the plerocercoid stage, has been reported from China, Japan, and other parts of the Far East. The parasites are variable in dimensions, and may measure 8 cm. to 36 cm. by 0.1 mm. to 12 mm. Similar parasites have been found in east Africa and British Guiana.

2. The parasite, Sparganum proliferum, has been reported from Japan and from the southern part of the United States. It is very variable in size and shape, but may attain 1 mm. to 12 mm. by 2.5 mm.

3. Sparganum baxteri measures 15 cm. in length by 1 mm. in breadth. Reported from German East Africa.

These larval tapeworms have been found under the peritoneum in the neighborhood of the kidneys, in the iliac fossæ, in the pleural cavity, in the urethra, beneath the conjunctiva, and in the subcutaneous connective tissue. The definitive host has been thought to be probably a carnivorous domestic animal, possibly the dog, cat, or pig. Others have conjectured that the carrier is more probably an aquatic bird or fish. Tumors, abscess, etc., have been found to be due to the presence of the parasite. When the worms invade the urinary passages, symptoms of stricture, pain, and hematuria are described. In cases of infection with Sparganum proliferum the parasites are found encysted in the subcutaneous tissue in large numbers. In the cases reported a history of previous intestinal teniasis was elicited. S. baxteri was found in an abscess in the thigh of a Masai.

Remedial Measures.—The treatment of this condition is essentially surgical. When the presence of Sparganum mansoni causes excrescences on the surface of the body, these should be removed by operation. When the cavities of the body are invaded, resulting in alarming symptoms, surgical relief should be sought if the cause of the disturbance can be diagnosed. As for the treatment of infection with S. proliferum, the cysts containing the parasite may be easily enucleated, the relief, as in the case of S. mansoni infection, being sought in surgical measures.

Preventive Measures.—Definite prophylactic measures cannot be outlined. In view of the probable method of transmission, and arguing from analogy, avoidance of uncooked food, especially fish, and care regarding contact with domestic animals should be enjoined.

Intestinal Teniasis.—There are numerous cestodes inhabiting the intestines of man which, while often reported from tropical and subtropical countries, have no claim to be regarded as in any way peculiar to warm climates. Several species, however, may be regarded as more or less directly tropical parasites. These will be taken up in order.

1. This parasite, Diplogonoporus grandis, has been twice found in Japan. The genital organs are disposed in double series, that is, two sets in each segment. The segments themselves are very short and broad. The genital pores open in longitudinal genital grooves. The uterus has only two prolongations to each side.

2. In Tænia africana the scolex is very small, quadrilateral, and unarmed, being provided with five suckers, one of which is apical. The segments are broader than they are long. The genital pores are irregularly alternate, being placed at the borders of the segments. The uterus has from fifteen to twenty regular prolongations. The eggs are spherical. It has been reported in German East Africa. I have a headless specimen of what is probably the same species from Rhodesia.

3. Tænia philippina measures from 80 to 100 cm. in length by 1 cm. maximum width. The head is cubical and unarmed. It has four suckers. The parasite was found by Hare in a prisoner in Bilibid prison in the Philippines.

4. In Tænia bremneri the segments measure about 28.6 by 8.5 mm. on an average. The head is not known. The worm was found in a Fullain woman in northern Nigeria.

5. In Tænia hominis the scolex measures 1.34 mm. in length by 2 mm. in breadth. The rostellum is unarmed. The edge of the suckers is

marked by a characteristic surrounding ridge. The entire length is stated to be 70 mm. Form of the genital organs not known. Reported from north of Persia.

6. In Hymenolepis nana the head is minute, the rostellum retractile, and the genital pores single, marginal, and unilateral. The species is very small, 5 to 45 mm. by 5 to 1 mm., the segments are broader than long. The uterus nearly fills the gravid segments. The scolex is armed with from twenty to thirty hooks. It has a pretty wide distribution throughout the tropical and subtropical regions of both hemispheres, and is not uncommon in certain sections of the southern portion of the United States.

7. In Hymenolepis diminuta the head is small, club-shaped, and has a rudimentary rostellum with four suckers, and is unarmed. The worm measures from 20 to 60 cm. in length and about 3.5 mm. in width at its widest point. The eggs are oval and measure about 60 μ in diameter. It is reported from South America, as well as various temperate countries.

8. In Davainea madagascariensis the rostellum is armed with two rows of hammer-shaped hooks, and the suckers are also armed with several rows of hooklets. The head is retractile, and the number of hooks about ninety. The worm is about 25 to 30 cm. by 1.5 mm., and the segments number from five to six hundred and contain calcareous corpuscles. The genital pores are unilateral. This species is reported from widely separated regions of the old and the new world tropics.

9. Davainea asiatica measures about 30 cm. in length and is composed of about seven hundred and fifty segments. The segments measure 0.16 mm. by 1.78 mm. The posterior margin of each segment overlaps the anterior margin of the following segments. The genital pores are unilateral. The uterus is broken up into sixty to seventy egg masses in each segment. Reported from north of Persia.

D. grandis is quite possibly due to eating uncooked fish. Infection with T. africana is thought to arise from eating the raw flesh of the zebu. The carrier of T. hominis is not known. H. nana is found in brown and black rats, mice, field mice, and dormice, as well as in man. White rats may be easily infected. The worm may also be directly conveyed from person to person, and autoreinfection may also take place. Blanchard has suggested that cockroaches may be responsible for the spread of D. madagascariensis.

Little is known of the pathogenesis of several of the foregoing parasites. H. nana invades the villi of the intestines and is said to injure the follicles of Lieberkühn. According to Grassi, it may bore deeply into the intestinal mucosa. As the worms are very numerous the effects may be severe. Abdominal pain, occasional diarrhea, epileptiform convulsions, headache, and various nervous symptoms are described. At autopsy the mucosa of the small intestine has been found to be tumefied and hyperemic.

Remedial Measures.—Male fern is almost a specific for tapeworms. Before the administration of the remedy the patient should be kept upon light diet for a couple of days, and preferably should fast for the last twenty-four hours. The bowels should be emptied by giving castor oil or Epsom salts. A good routine is: empty intestinal canal, castor oil the night before, no breakfast, anthelmintic on empty stomach.

The male fern may be given in capsules of the oleoresin, one 15-gr. (1.0-gm.) capsule being administered every ten minutes until half a dozen have been taken. Others give a single dram (4.0 gm.) dose. In women and children and in the case of men who are weakened from disease much smaller amounts should be used. Riesman warns against using castor oil for expelling the worm on account of the solubility of male fern in oils. He suggests calomel or magnesium citrate.

After treatment has been given, and the patient allowed to rest for an hour or so, another dose of salts should be administered and the resulting stool examined for parasites.

The parasites should be passed into a vessel containing warm water, otherwise the nervous organization of the tapeworm may result in its breaking in two and the head being left unpassed. To avoid this some recommend placing gauze on top of the water in the vessel to catch the worm and avoid traction. Under no circumstances should the worm be pulled from the rectum. If it breaks and the head is left in the bowel a salt enema will sometimes result in its expulsion. In case the head is not obtained the treatment should be repeated.

Extreme weakness, acute inflammation of the gastro-intestinal tract, grave cardiac or renal disease, and pregnancy are contra-indications to the use of male fern. Overdoses of the drug, even in healthy persons, may set up vomiting, diarrhea, collapse, blindness, jaundice, convulsions, etc.

As substitutes pumpkin seed, kamala, oil of filmaron, kousso, pomegranate root, or its alkaloid, pelletierin (tannate), may be given instead. Benzene is also recommended. Of these remedies pumpkin seed is the only one which can be safely given in pregnancy.

If necessary, tonics and supportive treatment should follow the expulsion of the worm, with iron and arsenic if anemia is present.

Preventive Measures.—Personal cleanliness, the careful cooking of fish and meat, care in the selection of drinking water, and avoidance of contact of natives or animals are all to be recommended. Houses should be kept clear of mice, rats, and other vermin, and such should be destroyed. In addition to infesting rats and mice in its adult stage, H. diminuta is said to live in its cysticercus stage in various insects (moths, earwigs, and beetles), and such insects should accordingly be destroyed in houses.

One should avoid sleeping in the same bed with a person infected with H. nana, and in endemic districts great care should be taken to keep all food out of the reach of mice and rats. Reisman makes the excellent suggestion that something regarding the methods of tapeworm infection and avoidance be put in school textbooks of physiology and hygiene.

DISTOMIASIS

Creighton Wellman

The number of flukes that occur in man in tropical countries is probably much larger than is at present known or suspected. In many warm countries it is difficult or impossible to secure necropsies, and many cases while alive do not come to European physicians. A number of species have, however, been reported from various parts of the tropical world.

Hepatic Distomiasis.—Several tropical flukes are found in the liver and bile ducts of man. The most important of these are the three following:

1. The fluke, Opisthorchis noverca, which is on the average 11 mm. by 2.5 mm. in size, seems to be rare in man, only having been found two or three times in India.
2. This fluke, Clonorchis sinensis, which averages about 12 mm. long by 3.3 broad, is found throughout the Orient, in India, Japan, Korea, Formosa, China, Tonkin, Mauritius, etc.
3. Clonorchis endemicus measures 6 to 13 mm. in length by 1.8 to 2 mm. in width. The eggs measure on the average about 26 μ by 15 μ and have a flattened operculum.

Opisthorchis sinensis inhabits the gall and bile ducts and is sometimes found in the pancreas and intestines. Occasionally the infection is very heavy, as high as 4,000 flukes having been found in one person. The presence of the flukes results in inflammation, dilatation, and sacculation of the bile ducts, and interstitial hepatitis, which is accompanied by atrophy and fatty degeneration of the liver cells. The functions of the liver are perverted by the pathological changes, and after a period of enlargement the organ becomes atrophic. The symptoms set up in extensive infection are chronic diarrhea, intermittent jaundice, and later cachexia accompanied by general edema. While the course of the disease is prolonged, yet it may prove fatal.

In cases of O. noverca the parasites appear in large numbers in the bile ducts, which are in consequence thickened and sacculated. C. endemicus also occurs in cats, dogs, pigs, as well as in man. The symptoms set up are diarrhea and enlargement of the liver.

Remedial Measures.—Removal of the patient from the endemic district or the institution of rigorous measures to prevent reinfection are logical procedures. Different drugs have been tried without success. In view of the beneficial effects reported from the use of salol in hepatic distomiasis of sheep, this remedy has been suggested in the present disease. Probably it should be tried in case the parasites are recognized by clinical signs or by ova in the feces. The various so-called cholagogues have also been recommended. Forchheimer suggests a cholecystotomy by means of which local treatment may be carried out.

Preventive Measures.—The mode of infection is unknown, and no specific measures can be advised. Cleanliness, the cooking of food, and the boiling of water are, of course, wise on general principles. Opisthorchis noverca is said to be found in the liver of dogs in India. C. endemicus infests cats and pigs besides dogs.

Intestinal Distomiasis.—A number of tropical trematodes are parasitic in the intestines of man. The most important of these are as follows:

1. The parasite, Gastrodiscus hominis, is a small fluke averaging about 6.5 mm. in length and 3.5 in breadth. The anterior portion is subcylindrical or roughly conical, while the posterior portion presents the appearance of a disk flattened and concave on its ventral surface. It is probably not a regular parasite of man, but is doubtless normally dependent upon some common mammal. It has been observed, however, in an Assamese from Gowhatly and an Indian in Tirhoot.

2. The parasite, Clonorchis watsoni, infests man in West Africa and is about 9 mm. by 4.5 in size. Manson believes it is not uncommon, especially in children. Probably similar African flukes, for example, the common Angolan antelope fluke described by Stiles and Goldberger under the name Wellmanius wellmani, will occasionally be reported in man.

3. Fasciolopsis buskii, the largest known trematode infesting man, averages about 30 mm. in length by 11 mm. broad. It has been reported from India, Siam, Sumatra, some sections of China (Canton), and Hong Kong.[1]

4. Fasciolopsis rathouisi is similar to F. buski, but averages only 17 mm. in length by 9.5 mm. broad. It occurs in China (where it is called Siau San Dzoong), and other parts of the far East.

5. Fasciolopsis füllerborni measures from 30 to 50 mm. long by 14 to 16 mm. broad. The eggs average 1 mm. in length by 0.073 mm. in width. This large parasite was found in the feces of a Hindoo in Hamburg.

---

[1] Apparently very similar to F. buski is an unnamed trematode described by Heanley. This fluke was found by a Chinese physician in a child in Hong Kong and is 2 inches in length by ½ inch in breadth. The cuticle is armed with spines and the genital organs closely resemble those of F. buski. The flukes were vomited by the patient.

6. Fascioletta ilocana measures from 4 to 6 mm. in length by 0.75 to 1.35 mm. in width. The eggs measure from 88.8 μ to 114.7 μ in length by 53.5 μ to 81.9 μ in breadth. This small species was discovered by Garrison in the feces of man in Luzon, Philippine Islands, in 1907.

7. The small parasite, Heterophyes heterophyes, which measures only about 1.5 by 5 mm., is probably of wide distribution, having been reported from such widely separated countries as Egypt and Japan.

Gastrodiscus hominis was found to be present in the cecum and colon in large numbers, the mucous membrane showing small red spots where the flukes had been attached by means of their suckers. Clonorchis watsoni was found by Watson, on postmortem, in the small intestine, the jejunum being full of flukes, some of them still adherent to the mucosa, which showed no distinct hemorrhages, but seemed somewhat congested. Before death the patient had numerous watery stools, deeply colored with bile and containing many adult flukes. Fasciolopsis buskii is parasitic in the duodenum of man, and its presence is said to cause fever (with temperature rising as high as 106° F.), bloody diarrhea, emaciation, lymphadenitis, edema, and other typhoid symptoms. It is in many instances fatal. The presence of F. rathouisi is said to cause diarrhea, light-colored stools of very offensive odor, tympanitis, wasting, and occasionally jaundice. Among the Chinese infection with this fluke is thought usually to cause death. Heterophyes heterophyes inhabits the small intestine, usually the central third of the jejunum, but does not seem to cause serious symptoms.

Remedial Measures.—In case the presence of intestinal flukes is suspected or indicated by the patient's passing adult worms or eggs, some anthelmintic, such as thymol, male fern, eucalyptus oil, or, as some recommend, chloroform, may be given while the patient is fasting. Such vermifuge should be preceded by a laxative and followed by a purge. Such treatment is specially successful in the case of Fasciolopsis infection, and the worms are expelled in large numbers. The purge employed may be Epsom salts, although when eucalyptus oil is employed the purgative had better be castor oil.

Preventive Measures.—The life cycles of G. hominis and C. watsoni are unknown, but probably uncooked food or infected water should be considered as dangerous. In the Orient animals should not be allowed near or about dwellings. The life history of other Paramphistomidæ suggests that avoidance of raw fruit and vegetables, especially water cress, and boiling water before drinking are measures to be adopted in endemic regions. F. buskii is said to be very common in South China in pigs. Living in proximity to these animals should be avoided and cooking food and boiling water, therefore, naturally occur to one as sensible prophylactic measures. Goddard believes that uncooked vegetables or raw fruit are involved in the spread of F. rathouisi. It is probably common to man and some domestic animal. Cleanliness, cooked food, boiled water, and keeping animals at a distance would seem wise measures against the spread of this species. As H. heterophyes seems to be common to several animals besides man (having been reported in dogs, foxes, kites, etc.), a rational prophylaxis would consist in the avoidance of these animals, and in cleanliness, especially the boiling of drinking water and cooking of food.

ENDEMIC HEMOPTYSIS

Creighton Wellman

Paragonimus westermanii measures about 7 mm. to 17 mm. by 4 mm. to 9 mm. The eggs are operculated and measure about 0.9 mm. by .06 mm. The conditions arising from its presence in the lungs are often seen in the Far East, and 2 cases from the Pacific Coast region, 1 reported by myself, have been observed. My own case came from Formosa, where the disease is endemic, a considerable part of the population being infected. It is probable that the disease will gain a foothold in the United States.

The infected patient usually has a chronic cough which is occasionally accompanied by attacks of hemoptysis and often a considerable degree of anemia. The hemoptysis may be slight or considerable. The lungs are tunneled by the parasites and infiltrated by their ova, and there is no hope of curing the condition. Mild cases, if reinfection is avoided, may recover spontaneously. Palliative and symptomatic treatment with general tonic measures are, therefore, all that can be aimed at.

Preventive Measures.—Sputum and other discharges containing ova should be burned. In endemic areas water should be boiled before drinking, and no uncooked food should be eaten. As domestic animals, such as cats, dogs, pigs, etc., harbor the parasite, both in the Orient and here in the United States as well, association with these should be avoided.

SCHISTOSOMIASIS

Creighton Wellman

There are three species of Schistosoma known to affect man in tropical and subtropical regions. The diseases arising from the presence in the tissues of these different worms will be considered separately.

Endemic Hematuria.—This disease is due to Schistosoma hæmatobium and is found in Africa, Mauritius, and the near East. S. hœmatobium is flattened in contour, and the male measures 11 mm. to 15 mm.

by 0.9 mm. to 1.1 mm. The female is rounded and measures about 18 mm. to 20 mm. by 0.2 mm. to 0.3 mm. The eggs are oval and have a terminal spine. They measure about 0.12 mm. to 0.2 mm. by 0.05 mm. to 0.07 mm. The integument of the male is tuberculated. The parasites are found in the portal vein and its branches, the veins of the bladder, and of other pelvic organs. The female deposits her eggs in the smaller veins, especially in the walls of the bladder, and these eggs escape through the mucous membrane into the lumen of the bladder and are passed with the urine. Other organs, notably the liver and rectum, are also invaded.

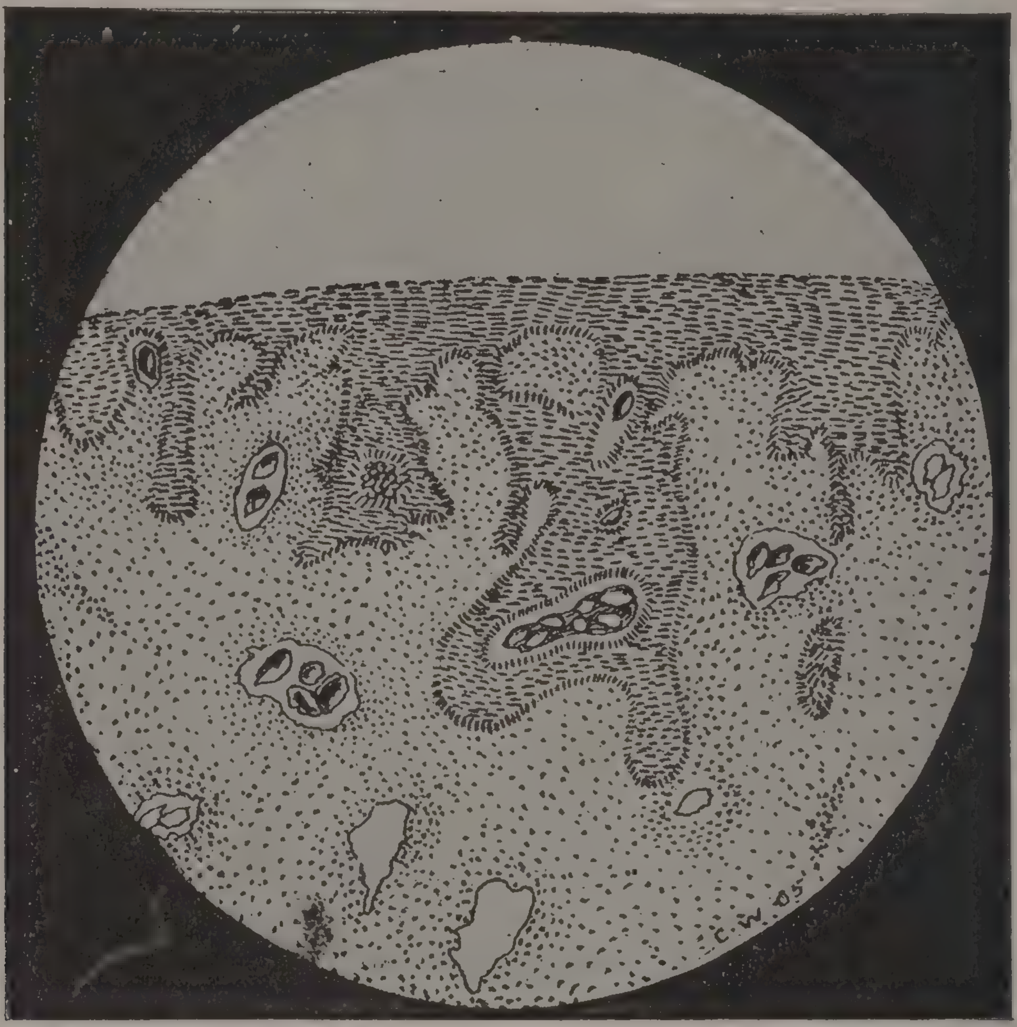


Fig. 1.—Schistosomal Papilloma of the Rectum. Section showing ova of S. hæmatobium in situ. (After Wellman.)

The conditions set up by the presence of the parasite are papillomatous-like growths in the mucous membrane of the bladder, ulcers, thickening of the bladder walls, etc. Some blood is usually passed with the urine, generally to be seen in only the last few drops passed. Sometimes, however, the hemorrhage is extensive and the entire contents of the bladder may be colored with blood. Urinary fistulæ, calculi, urethral stricture, etc., may be produced. In many cases, however, the presence of the parasite gives rise to little discomfort, and most of the long series of cases seen by me in West Africa were of a mild type.

Remedial Measures.—No specific treatment can be attempted. Some authors have advocated the use of male fern, methylene-blue, etc., but results do not appear to be encouraging. As Manson observes, the treatment resolves itself into that of chronic cystitis. Urotropin in doses of 5 gr. (0.30 gm.) three times a day may be tried.

In a long series of cases treated by me in West Africa I found excellent results from the exhibition of hyoscyamus, probably on account of its sedative effect in the prevention of vesical tenesmus. Where the hematuria is excessive, adrenalin may be tried to control it and sedatives may be indicated for the pain which is sometimes present.

Irrigation of the bladder with a boric acid solution (1 per cent), followed by 1:5,000 solution of nitrate of silver, has been very beneficial in my experience.

Calculi, fistulæ, stricture, etc., should be dealt with surgically. In severe cases rest in bed and tonics are indicated. The patient should be encouraged to take much liquid. Nourishing, non-irritating food should be recommended, stimulants and spices being avoided. Overexertion and other excesses, chill, and all physiological depression should be guarded against.

I have found that the administration of cod-liver oil is usually of great benefit in this chronic and exhaustive disease.

Preventive Measures.—In endemic areas one should avoid bathing in streams, pools, and public baths. The urine and feces of patients should be destroyed or disinfected. Natives should be specially instructed against urinating into streams or other collections of water. All food and drinking water should be cooked or filtered, and the source of supply furnishing water for bathing and other domestic purposes should be carefully inspected.

Intestinal Schistosomiasis.—This disease occurs in Africa and tropical America, including the West Indies, and is due to infection with Schistosoma mansoni. The male measures about 12 mm. by 0.44 mm., and the female about 14.5 mm. by 0.16 mm. The eggs measure 149 μ by 62 μ and have a lateral spine.

This parasite is found principally in the mesenteric veins. The eggs being deposited in the submucosa of the intestines, especially the rectum, give rise to infiltration and papillomatoid growths, which are accompanied by various symptoms. Diarrhea and dysentery are most frequently seen. Occasionally other organs, notably the lungs and the liver, may also be affected. In case of such latter involvement a peculiar fibroid pneumonia and cirrhosis of the liver are found.

Remedial Measures.—As in the case of endemic hematuria, no specific treatment can be attempted, and palliative measures are the most that can be hoped for.

In case infiltrated tumors of the rectum and other intestines can be felt, these should be treated surgically. Enemata of quinin and sodium hypochlorid (1:100) have been recommended when there are ulcers low down in the bowels.

Preventive Measures.—These are the same as those for the prevention of endemic hematuria.

Katayama Disease.—This affection is caused by a third species, which probably has a wide distribution in the Orient (Schistosoma japonica). The male measures 9 mm. to 12 mm. by 0.5 mm., and the female 12 mm. by 0.4 mm. The eggs measure 70 μ to 75 μ by 45 μ to 55 μ, and are smooth and without any prominent spine. The integument of the male is not tuberculated.

The adult parasites seem to inhabit the smaller blood-vessels of the mesentery, and the ova are found in the mucous and submucous coats of the intestine principally. There may be, however, involvement of the liver, mesenteric glands, pancreas, and almost every other organ of the body, including the brain. The bladder has not been shown to be affected. When the brain is involved, headache, vertigo, nervous manifestations, jacksonian epilepsy, and paralysis may all appear.

Remedial Measures.—As in the case preceding, no specific treatment is available.

Preventive Measures.—Since it has been shown that the cat is a carrier of S. japonica, destruction of these animals in endemic districts should be insisted on. Avoidance of uncooked food and impure water is also to be considered.

FILARIASIS

William B. Wherry

The failing interest of the medical profession in the rôle played by animals in the production of disease was most successfully revived when the white man invaded the tropics. One by one the causes or means of transmission of many hitherto obscure diseases were found to fall into this group. The precise information concerning the life history of these parasites, the acquisition of which necessarily precedes rational prophylaxis, was largely the result of the investigations of zoölogists. But in many instances the pages record the labors of the physician in the far parts of the earth.

Filaria Bancrofti—*Prophylaxis.*—Through the brilliant experiments of Manson in China, Bancroft in Australia, Low in the West Indies, and James in India, it has been definitely established that certain mosquitoes act as immediate hosts for the embryos of F. bancrofti. The embryos F. nocturna, circulating with the peripheral blood of the human host, are taken up by various species of Culex and Anopheles. In the tissues of these insects they develop and undergo a metamorphosis, which necessarily precedes their reintroduction into a human host. The most widely distributed of these mosquitoes is Culex fatigans, var. skusii, a voracious night feeder, which acts as intermediate host in Asia, Australia, the West Indies, and elsewhere. James showed that Anopheles rossi, Culex microanulatus, and Culex albopictus were favorable hosts in India. According to Annett, Dutton, and Elliott, F. nocturna undergoes complete metamorphosis in Anopheles costalis in West Africa. The theory that infected mosquitoes reinoculate the human host during a subsequent feeding received support from the domain of comparative pathology. Grassi and Noè were able to produce infection in muzzled dogs which had been subjected to the bites of mosquitoes infected with the larvæ of Filaria immitis. Hence, prophylaxis here coincides with that against malaria. Protecting the mosquitoes against infection, by screening human hosts, is almost as important as culicidal measures.

Treatment.—The treatment of the various lesions resulting from the obstruction of the lymphatics has perhaps some hope of now being transferred from the surgical side to the medical. Attempts to kill the parasites with thymol, extractum felicis, methylene-blue, benzoic and boric acids, sodium benzoate, picric-nitrate of potash, etc., have been abandoned as failures. The usual termination of hematochyluria was suicide; of varicose glands, recurrent lymphangitis, often followed by elephantiasis.

McDill and the writer treated a case of hematochyluria with the X-rays and, at the suggestion of Musgrave, cinchonized her before applying the rays. This patient recovered. The embryos disappeared from her blood, and she was in perfect health four years later.

Wellman and von Adelung tried this method with apparent success, but the latter reported on its failure later on.

It appears, however, that recent arsenical preparations kill the adult worms. This means much, for the early destruction of the parasites promises relief from many hopeless conditions and from the surgical intervention which is so often the last resort. O'Brien killed off the embryos F. nocturna from the circulation of a Cingalese with atoxyl; and, as they did not reappear during a year's observation, it is presumable that the parent worms were killed. This case received intravenous injections of 5 minims, gradually increased to 40 minims, of a 5 per cent solution in sterile saline. It was given at first every three or four days, and later every day, the whole treatment lasting about six weeks.

Reichmann gave 0.3 gm. "Ehrlich-Hatta" to a patient suffering from pleurisy with an undetermined filaria-like worm in the pleural exudate. This was followed by recovery.

Pilcher and Pilcher treated a white female, thirty-five years old, who developed chyluria four years after leaving the tropics. Chylous urine was secreted from the left ureter only. She was given 0.6 gm. "606" hypodermically without any marked constitutional derangement. The filarial embryos, which appeared only at night in the peripheral circulation, and hence were probably the embryos of F. bancrofti, disappeared within twenty-four hours after the injection. Between the sixty-eighth and seventy-second hours following the treatment the urine became markedly chylous and contained a large number of dead embryos and threadlike adult parasites.

Werner (1911) reports a failure after the intravenous injection of 0.5 gm. arsphenamin. The F. nocturna were not diminished in numbers three days after treatment.

For surgical technic in connection with filariasis, the reader is referred to the articles of Charles, Werner, Stokes, and Lanz.

Dracunculus Medinensis (Guinea Worm, Medina Worm, the Worm of Pharaoh, etc.)—Prophylaxis.—The idea that infection with the guinea worm is acquired by drinking polluted waters can be traced back to antiquity. O'Donovan, who explored the Merv oasis, records the statement of a Khan of the Turcomans, "that anyone who drinks the stagnant water of the rain pools, or that of the tanks of the caravanserais (in southern Bokhara), is certain to take into his system the egg of the *rishté,* which will then infallibly develop itself." Several observations point to the incubation period in man being from eight months to a year.

The experiments of Fedschenko in Turkestan, which have been confirmed by Manson and Blanchard, point to certain fresh water Cyclops (Cyclops quadricornis, strenuus, viridis, bicuspidatus) as the intermediate host. The filarial embryos escaping into water pass through the integument and into the body cavity of the Cyclops. There they undergo a metamorphosis somewhat similar to that of F. nocturna in the mosquito. Wenyon confirmed the observation of Leiper, that the addition of very dilute hydrochloric acid caused the completely metamorphosed embryo to desert the Cyclops—thus simulating the probable course of events in the human stomach. These experiments make it almost certain that infection is acquired through drinking water. However, as Plehn was apparently successful in infecting a monkey by feeding the undeveloped larvæ, one should guard against more direct methods of infection, as well as adhering to the use of boiled or carefully filtered water.

Treatment.—The ancient method of gradually extracting the untreated worm by winding it on a split stick should be avoided. Accidental rupture of the parasite is followed by serious results—intense inflammation and suppuration, often followed by bone necrosis, septicemia, or contractions and ankyloses.

In the endemic regions natives who make a vocation of extracting the worm acquire great skill. With their small light hands they knead the worm toward the opening and seldom break it. According to Maclean, the practice of douching the part with cold water to aid extraction was universal in the endemic regions in India. There is no doubt that this practice hastens the emptying of the worm's uterus, and so minimizes the dangers incident to extraction. Manson recommends the following procedure: the site occupied by the parasite should be douched with cold water three or four times a day, and in the meantime protected by dressings wet with water. As a result of this stimulation the uterus empties itself more rapidly. Complete evacuation is assured when the worm no longer reacts to the water test. Now extraction is comparatively easy.

However, the method of Emily is perhaps the best and safest, and its value has been amply confirmed. If the worm is protruding, a 1:1,000 solution of bichlorid of mercury is injected, by means of a fine syringe, into its body; twenty-four hours later it may be extracted with ease. If the worm is not visible, a few drops of the same solution are injected as near it as possible. The worm dies and is absorbed, or may then be extracted with the aid of a knife.

Filaria Loa.—It is not known just how infection with F. loa takes place. The weight of evidence points to F. diurna as the embryo of F. loa. This is supported by Huffman, who studied the embryos extracted from a live loa. On the assumption that the embryos of F. loa circulate in the daytime, Manson has suggested that some day-biting insect, such as the Mangrove fly (Chrysops dimidiatus), must act as intermediate host.

It seems certain that F. diurna must be a different embryo from F. nocturna, for Annett, Dutton, and Elliott found that the latter developed in Anopheles costalis, while the former did not.

Brumpt did not succeed in developing F. diurna in a species of Glossina, but the list of biting flies in the endemic region has by no means been exhausted. That the disease is perhaps only acquired in "the bush" may follow from information acquired by the writer from Dr. Emil Blunden, long a missionary at Batanga, German Cameroon, West Africa. He felt that there was much a priori evidence that loa infection followed the bites of certain flies, and cited as evidence that, according to his observations, when women accompanied their husbands on itinerary expeditions, they were severely bitten by flies, and that all these women who travel about much become infected with loa. Their husbands, who lead a more active life, are less severely bitten, and often escape infection. Further, the wives of government officials who stay at home escape infection.

Treatment.—Usually the presence of the loa is not suspected until it is seen making its way beneath the skin of the body or extremities, or under the conjunctiva. Its passage beneath the conjunctiva is usually accompanied by a stinging pain and slight edema. Persons harboring the loa usually complain of the repeated appearance of single or multiple swellings on various parts of the body—especially the extremities. These are the so-called "Calabar swellings." Manson has brought forward evidence to show that these are due to the subcutaneous discharge of the embryos of the loa—the resulting edema being analogous to that following the rupture of the Medina worm. In the case of the loa, however, the swelling is transitory. Taken altogether, the symptoms hardly call for medical treatment. However, it is customary, particularly in the case of neurasthenic whites, to remove the parasites one after another, as opportunity offers. When one appears beneath the skin or conjunctiva, it is seized and held by forceps while cocain is applied. Then it is cut down upon and removed. This should be done without rupturing the parasites, for rupture is followed by intense local edema which, in the case of the eye, is uncomfortable, and, though not dangerous, requires treatment for three or four days.

Perhaps rupture of the male is not followed by edema. Dr. Blunden ruptured a loa while removing it from the conjunctiva, and there followed an intense edema. This worm was one of the female loas described by Huffman and the writer.

Filaria Perstans.—Wellman found what he believed to be developmental forms of this filaria in ticks (Ornithodorus moubata) fed on infected blood. His experiments were performed with ticks bred from eggs and conducted more carefully than those of Feldman. Fülleborn was unable to confirm these observations, and also had failures with Stegomyia fasciata, Culex pipiens, and Pulex irritans. He refers to the work of Noè, who had reported the metamorphosis of the Filaria Grassi of dogs in a tick.

OTHER NEMATODE INFECTIONS

Creighton Wellman

Revised by Samuel T. Darling

Strongyloides Stercoralis.—This parasite has a geographical distribution closely coinciding with that of hookworm. The worm, Strongyloides stercoralis, is heterogeneous in its development and several stages, consisting of two phases—a parasitic generation and a free-living form—which were formerly thought to be different parasites, are found in man. The heterogamy of Strongyloides stercoralis has resulted in much confusion regarding its name. It is seen in two forms: (1) the parasitic generation measures about 2.2 mm. by 0.034 mm. The cuticle is finely striated. The mouth has four lips, the esophagus is cylindrical and about one-third the length of the body. The anus opens near the posterior extremity, the vulva at the posterior third of the body. The eggs average about 0.055 mm. by 0.032 mm. This generation is either parthenogenetic or hermaphroditic. (2) The free living generation is sexually differentiated. The body is smooth and the mouth has four lips. The esophagus is short with a double rhabdoid dilatation. The females measure about 1.5 mm. by 0.08 mm., the males 1 mm. by 0.05 mm., and the ova 0.07 by

0.045 mm. Most specimens measured from cases seen in Panama were slightly larger than these. In specimens of feces or in cultures of the same there is among natives of the tropics a predominance of rhabditiform larvæ which develop in the soil into the sexually differentiated generation. Among natives of colder climates there is a predominance of larvæ somewhat longer and slenderer, known as filariform. Rarely ova are found in the stools.

The infection arises through invasion of the skin by the filariform larvæ either of the direct parasitic generation or of the filariform larvæ of the sexually differentiated generation. These larvæ develop into the female mother worms which invade the mucosa and sometimes the submucosa of the stomach and upper part of the small intestine. Here in the epithelium of the crypts the embryos are placed, and some are deposited free in the lumen of the bowel. Certain tissue reactions take place such as proliferation of round and connective tissue cells in those portions of the mucosa invaded by the worms.

Portals of entry for microörganisms must be made by the mother worm and her embryos, although no case of generalized infection is known to have arisen in this way.

Symptoms.—"Cochin China diarrhea" was formerly thought to have been caused by this nematode but it is not believed at present that the worms can provoke diarrhea. Among over one hundred cases of this infection studied at Ancon hospital there were no cases in which diarrhea was a symptom of any note. The stools of animals infected with other species of the genus are well formed. On the other hand, in animals and in man infected with this class of nematodes anemia has been observed, although it has been impossible to say that it was entirely due to the activities of the nematode.

Askanazy and the writer have found that the female mother worm and her embryos which are imbedded in the mucosa contain fat droplets and Askanazy believed that the mother worm obtained sustenance in the latter location. While nothing definite is known about the possible pathogenic activities of this nematode, it should probably be regarded as a possible cause of anemia and as a portal of entry for microörganisms, even the tubercle bacillus.

Remedial Measures.—In view of the fact that the worms burrow into the mucous membrane of the intestine, treatment is unsatisfactory. Various anthelmintics have been used, but without much success. Chenopodium has been recommended by several writers, but in the hands of the writer it has proved of no value.

Carbon tetrachlorid is more active in the upper part of the intestinal tract and may be given a trial (see article on Hookworm).

Preventive Measures.—The prophylaxis should be the same as that against hookworm.

Trichocephaliasis.—Trichuris trichiura, commonly known as whipworm, is very common in warm regions, in some districts over half the inhabitants being infected. The worm measures from 40 to 45 mm. (male), and from 45 to 50 mm. (female) in length. The eggs are longly oval, brownish in color, thick-shelled, with a clear plug at each end, and measure about 50 μ by 23 μ.

The worms are found in the cecum, more rarely in the ascending colon and appendix. The effects on the host are not thought to be serious, although many authors consider that, as the anterior extremity of the worms is often found buried in the intestinal mucosa, the presence of the worms may facilitate the entry of secondary bacterial invaders, and lead to intestinal disturbances, appendicitis, etc. The received ideas as to the innocuousness of T. trichiura would seem to need revision since the published fatal cases of Musgrave, Clegg and Polk, and of Jamieson and Lauder. The pathology of such cases appears to consist of a severe secondary anemia.

Remedial Measures.—Anthelmintics may be tried, although it seems to be the consensus of opinion among those who have studied the parasite that it is impossible to expel it with any degree of thoroughness. Musgrave and others recommend benzene irrigations, 1 to 4 drams (15 to 60 c.c.) to the quart of water. Riesman considers that, "unless it can be definitely proved that the worms are causing disturbances, it is best not to persist in attempts at their removal."

Preventive Measures.—Cleanliness, a pure water supply (or at least boiling or filtering of suspicious water), and the avoidance of pets which harbor the worm are all to be kept in mind.

Strongylosis.—Several small worms belonging to the genera Strongylus and Trichostrongylus have been reported from the tropics as occurring in man.

1. In Strongylus subtilis the males measure about 4 to 5 mm. by 0.09-0.07 mm. The female measures 5.6 to 7 mm. by 0.09 mm. The eggs are oval, with thin shells, and resemble hookworm ova.
2. In Strongylus gibsoni the male measures 21 mm. in length and has the anus 0.2 mm. and the vulva 0.5 mm. from the tip of the tail. The eggs measure 110 μ by 53 μ.
3. In Trichostrongylus instabilis the male measures 4 to 5 mm. in length by about 0.08 mm. in breadth at its greatest diameter. The female measures 5 to 6 mm. in length by about 0.09 in thickness. The anus is about 0.07 mm. and the vulva about 1.2 mm. in front of the posterior extremity. The eggs measure 72 to 80 μ by 40 to 43 μ. The life history is unknown.
4. In Trichostrongylus probolurus the male measures 4.5 to 5.5 mm. long by 0.08 mm. at its greatest diameter, and the female 4.5 to 6 mm. by

1.15, the anus being 0.05 mm. and the vulva 1.25 mm. in front of the posterior extremity. The eggs measure 76 to 80 μ by 43 to 46 μ. The life history is unknown.

5. In Trichostrongylus vitrinus the male measures 4 to 5.5 mm. long by .085 mm. thick at its greatest diameter. The female measures 5 to 6.5 mm. long, while the vulva is 1.15 to 1.25 mm. in front of the posterior extremity. The eggs measure 84 to 90 μ by 46 to 50 μ. The life history is unknown.

S. subtilis occurs in camels and men in Egypt and the Orient. T. instabilis and T. probolurus are normally parasites of sheep, antelopes, and dromedaries, the former occurring also in monkeys. Possibly the undetermined nematode embryo found by myself in the blood-stream of a West African negro is to be referred to some such worm as the above. In such an event the life history probably resembles that of hookworm.

No particular ill effects have been noticed from the presence of these parasites in the intestine.

Remedial Measures.—The worms are delicate and probably any vermifuge followed by a purgative would expel them.

Œsophagostomiasis.—Several species of Œsophagostoma have been found in man in the tropics, as follows:

1. In Œsophagostoma brumpti the female only is known and measures 8.5 to 10.2 mm. by 0.295 to 0.325 mm. Reported from West Africa.

2. The worm, Œsophagostoma stephanostomum var. thomasi, from South America is similar in its structural characters and habits to the foregoing. It has been suggested that some of the forms of tropical anemia in Central and South America may be due to such worms as this. The species under discussion has been studied carefully and causes cystic nodules in the walls of the intestine. Romanovitch has reported septicemia in a monkey caused by œsophagostomiasis.

3. Œsophagostoma apiostomum resembles Necator americanus rather closely in size and general macroscopic appearance, but lacks the characteristic crook at the anterior end of the body, and possesses a terminal mouth which is surrounded by setæ which protrude through its aperture.

Leiper has noted extensive diarrhea and scouring in many of the lower animals caused by various species of Œsophagostoma, but points out that in the tropics the cysts and lesions of the epithelium associated with the presence of these worms in man might easily be overlooked. The worms encyst themselves in the walls of the cecum and colon and should be thought of when an unexplained dysentery with anemia is present.

Remedial Measures.—There is no hope of expelling the two species first mentioned by means of a vermifuge. The last-named worm will probably be confused with hookworm, but if recognized can evidently be expelled with thymol.

Other Nematodes of Doubtful Pathogenesis.—In addition to the foregoing there are a few worms parasitic in man which have some claim to be considered tropical parasites. These are Tridontophorus diminutus, Lagochilascaris minor, and Physaloptera mordens. Owing to space considerations, they will not be considered in detail here.

Gnathostoma Siamense.—The female worm only is known and measures 9 mm. by 1 mm. The anterior third of the body is set with spines. This parasite was first found in the breast of a young Siamese woman. The patient had a hard painful swelling in her breast accompanied by fever. On the skin appeared nodules the size of a bean, from one of which the worm was removed. Two other similar cases have since been reported. Similar species live in the stomach of cats, dogs, pigs, oxen, and monkeys in the Orient and in South America.

The treatment is surgical.

Leeches

A number of species of these animals attack man in tropical countries. Most of them are distributed in collections of water, particularly pools with stagnant or swampy water visited by animals.

A troublesome form is encountered in damp forests and in wet sylvan glades. These are smaller than those found in pools and they climb up the vegetation for a sufficient distance to attach themselves to passing animals and man. A few of the most important tropical species are Limnatis nilotica, L. mysomelas, L. granulosa, Hæmadipsa ceylonica, etc.

L. nilotica is usually taken into the mouth with drinking water, or attacks persons bathing. It has been observed in the pharynx, larynx, esophagus, nasal cavities, vagina, and conjunctiva. H. ceylonica clings to leaves or twigs and springs upon passing men or animals. Its bites are said to be extremely liable to become painful and dangerous ulcers. Death has been known to result from the attacks of large numbers of these parasites. It should not be forgotten that leeches are the hosts of trypanosomes and other parasites, and their bite may introduce these into man as well as into animals. Numerous undescribed species occur in various parts of the tropics.

Remedial Measures.—When the animals were in the nares Manson succeeded in removing them by attaching, through a speculum, a spring forceps to their hinder end, and afterward injecting salt and water. They should be carefully looked for in the mucous orifices of the body when their presence is suspected. When attacking the skin they should be promptly removed and an antiseptic applied.

Preventive Measures.—It is sometimes difficult to protect one's self from attack, as some species are able to force their way even under close-fitting garments. Manson recommends that when passing through jungle lands in which leeches abound one should have the feet and legs protected. Woolen puttees which are wrapped around the leg are effective in keeping leeches out. It is helpful to wear stout socks which may be brought out over the folded-in trousers ends. Care in the selection of drinking water is, of course, an obvious precaution.

REFERENCE

Bavay. Compt. rend. Acad. d. sc., lxxxiii, 694, 1876; lxxxiv, 266, 1877.

CHAPTER XLIV

AFFECTIONS DUE TO POISONOUS REPTILES, FISH, ARTHROPODS (INCLUDING MYIASIS), AND PLANTS

CREIGHTON WELLMAN

REVISED BY SAMUEL T. DARLING

SNAKE POISONING

Venomous snakes grow to a larger size in the tropics and their bites are dangerous and often fatal. Two families and a subfamily are generally recognized, Viperidæ, Colubridæ and Hydrophinæ.

The family Viperidæ or vipers contains the well-known copperhead, water moccasin, bush master (surucucú of Brazil), Urutú, fer-de-lance and rattlesnakes. These are the pit vipers, so-called because of the deep depression on each side between the eye and the nostril. The family also includes the true vipers, such as Russell's viper of India, the puff adder and horned viper of Africa.

The other family Colubridæ is represented in America by the coral and harlequin snakes; in Asia by the cobra and the hamadryad and kraits; in Africa by the Egyptian asp; in Australia by the black and brown snakes; and in Europe by the European viper.

The water snakes of a venomous nature are included in the subfamily Hydrophinæ containing the sea snake of the Indian Ocean and another species living in fresh waters of the Philippines.

The snake in striking coils and thrusts its body forward about one-half its length. The head is bent backward and the jaws widely opened. The maxilla is rotated forward and the fangs erected. In biting, the closure of the jaws by muscular contraction squeezes venom out into the wound made by the fangs, for the poison gland is enclosed by the temporal muscle.

The venom is secreted by what is histologically a serous gland homologous with the parotid. It is clear or slightly milky and of a straw or slightly greenish tint; very complex in composition and in its effects on animals. Its toxicity depends on the species of snake, the amount injected and the condition of snake and also on the species and age of the animal bitten.

Snake venom varies in composition with the different species of snakes, but the following substances determined by their physiological effects on animals have been noted: (*a*) neurotoxins, having specific effects on the respiratory and vasomotor centers, and others acting on muscle-nerve-end organs; (*b*) agglutinins, a fibrin ferment, an antifibrin ferment, a proteolytic ferment and agglutinins; (*c*) various cytolysins active against erythrocytes, leukocytes, endothelial cells and nerve cells besides antibactericidal and other principles.

The effects of the venom of a particular snake will depend on the properties of the constituent substances in the venom. The toxic powers of the venom of Colubridæ are greater than those of the Viperidæ and they differ in character.

Symptoms of Colubrine Poisoning
(*the Cobra, Naja tripudians*)

Intense pain at seat of bite, local burning extending up the arm or leg to body, local edema and congestion.

Intoxication, prostration, muscular weakness and paralysis, cold sweats, salivation becoming profuse, nausea, vomiting, somnolence, rapid respiration. Marked paralysis of tongue and pharynx, inability to speak. Death due to failure of respiration.

Symptoms like acute bulbar paralysis.

Symptoms of Viperine Poisoning
(*Russell's viper, Vipera russellii*)

Painful swelling about the bite, rapidly spreading. Excitation, thready pulse, rapid respirations and great prostration, stupor, cold sweat, convulsions and circulatory failure.

Blood is fluid at autopsy. (After poisoning by the rattlesnake.)

The diamond rattler causes local pain, hemorrhage, edema, ecchymoses at the bite with sweating, prostration, vomiting, diarrhea, dyspnea and collapse.

General and Specific Treatment.—A number of specific sera have been prepared and have proved of great value in reducing the mortality from snake poisoning. This is particularly true in Brazil where the Instituto de Butantan in the State of São Paulo prepares sera active against some of the more numerous venomous snakes of that and adjoining states.

As these sera are strictly specific it is necessary to know the species of snake that has bitten the patient. In Brazil this is usually known and in every village and on many plantations sera are kept on hand for emergency requirements. Sera must be given shortly after the receipt of the bite to be efficacious, and they must be given in proportion to the amount of venom injected. If the circumstances demand urgent action, the serum may be given intravenously, otherwise it may be administered subcutaneously or intramuscularly. The patient must be carefully watched and additional serum given if the symptoms demand it.

Non-Specific Treatment.—When specific sera are not available, it is recommended to:

Prevent absorption or flow of lymph and blood away from the injured spot or bitten area.

Apply medicaments locally to neutralize or destroy the venom.

Aid in the removal of excreted venom.

When a limb has been bitten, two or three tourniquets should be applied proximal to the bite, and the tissues near the bite should be freely incised along the course of the blood-vessels and lymphatics. The ligature must not be applied continuously for too long a period, but may be loosened for a few seconds and reapplied.

A strong solution of potassium permanganate may be applied to the tissues using a small lancet to reach the deeper parts.[1] On the supposition that the venom is excreted from the gastric mucosa gastric lavage has been recommended.

Artificial respiration should be maintained when there is respiratory failure, and very large doses of strychnin may be used hypodermically as a stimulant. Müller recommends 1/10 gr. every fifteen minutes until urgent symptoms have disappeared.

Many cases of snake bite do not prove fatal, for much depends on the dose of venom received and the age of the person. It is impossible to know what value some of the general non-specific measures possess in treatment.[2]

Prophylaxis.—The best preventive against snake bites is the use of the long pigskin or other leather putties. They should be used by all persons who have to go about in the trails or bush where snakes are likely to be. Tight screen or other doors and absence of holes or spaces in walls and floors are necessary to keep snakes out of the house in the dry season in some localities. Grass cutting for some distance around quarters will keep snakes from coming near the house as well as reduce the numbers of mosquitoes and sand-flies.

OTHER POISONOUS VERTEBRATES

Batrachia and Lacertilia.—Among the Batrachia may be mentioned the salamanders (for example, Cryptobranchus japonicus), and the toads (for example, Bufo agua). Of the Lacertilia there is only one important poisonous genus, namely, Heloderma (H. horridum) from tropical America.

Salamanderin and the poison of Heloderma are said to act much like viper venom. Several toxic substances have been studied from toads: bufotalin, a heart poison; bufotein, a nerve poison, and phrynolysin, an erythrolytic substance. Here may be mentioned bufagin, from the so-called parotid glands of Bufo agua, identical or closely allied to epinephrin.

---

[1] In India it is common practice to rub the crystals of permanganate directly into the wound.—Editor.

[2] The administration of whisky as often practiced in the United States is of very dubious value and has probably caused death in some cases.—Editor.

Remedial Measures.—In the absence of specific antisera the poisons of the above are best combated by the general measures given for snake bite.

POISONOUS FISH

Fish poisoning may be brought about in a variety of ways. There are fish (Muræna and Tetrodon) which poison by means of their bite. These are found in tropical waters in the Atlantic, the Mediterranean, and the China Sea. The poison is secreted by an apparatus lying within the mouth and the venom flows into the wound caused by the bite.

Another group possesses an external poison apparatus either closed and requiring rupture or having an external opening through which the venom is ejected, or passively expelled by the erection of a spine or barb. The venom glands are located beneath certain spines or barbs in relation with the dorsal or anal fins, or the gill covers.

The venom is probably protective in nature and cases of poisoning arise in man in handling fish or by frequenting waters in which the venomous species abound. Some species undoubtedly attack bathers, while children and adults wading for shellfish along the shores may by stepping on the fish or otherwise coming in contact with the poison apparatus receive a bite. The venom appears to vary in composition and in its effect on tissues.

The symptoms of poisoning are usually those of a stinging pain at the wound or point of entrance of the barb or spine. There is an intense burning or stabbing pain which rapidly extends up the limb or radiates outward from the wound. The part becomes numb or the pain increases to an extreme severity. Vomiting is common and when the pain is agonizing in character there is usually great prostration, delirium and syncope. In the milder cases the symptoms wear off in a few hours, but in those of a more severe character the parts become much swollen and tissue necrosis follows after several days of illness.

Thalassophryne of the Bay of Panama will attack swimmers and the symptoms are so acutely painful that prostration and drowning may easily occur. Indeed there can be no doubt that these fish are the cause of drowning in a good many instances.

The *sting rays* are armed with a spined tail. Usually there is an area of redness and swelling in the skin surrounding the place stung. Pain and swelling develop rapidly; these are localized, however, and while disagreeable are not serious. A species found in Japanese waters causes severe inflammation and gangrene in the parts around the wound.

Treatment.—Pain is intense and will require injections of morphia. Stimulants such as caffein sodium benzoate (gr. ii hypodermically) may be required if syncope occurs.

Preventive Measures.—Great care should be taken to identify all tropical fish before eating. Among the natives some individuals are immune to the ill effects of eating these fish, so caution should be displayed in accepting the assurance of guides, etc., as to the harmlessness of any certain variety of fish.

Poisonous Mammals.—The Australian Ornithorhynchus paradoxus, by means of poisoned spurs, may inflict bad wounds. The poison is stated to be much like snake venom in its action, although much milder.

The bites of carnivora are to be considered as open, infected wounds.

Remedial Measures.—The treatment of Ornithorhynchus bites consists of the general measures used in cases of snake bite. The ill effects following the bites of carnivorous animals may require treatment for general septic infection or even rabies.

VENOMOUS ARTHROPODS

Spider, Centipede and Scorpion Poisoning

Spider Bite.—Some large and particularly venomous species of spiders are found in the tropics. The more important species are Lathrodectes (Latrodectes?), the red back spider of Australia, L. hasselti, L. scelio of New Zealand, and L. mactans of South America and elsewhere. Various species of Theridium, Theraphosa, Glyptocranium and others are responsible for many serious cases of poisoning.

Some species are found in the field, others in vineyards. L. hasselti hides under the seats of water closets in Australia and the Orient and oftentimes painful bites on the genitalia are produced.

The effects are in general like those following the bites of scorpions. There is very severe local pain which passes into paresthesias and paresis. There is sweating, extension of pain and prostration, sometimes with tetanoid state. Death sometimes ensues after a typhoidal state. Local edema, hemorrhages and necroses may occur after the bite of certain species.

Treatment.—Incision and the application of a strong solution of potassium permanganate to the tissue around the bite is indicated. Pain and circulatory and respiratory failure should be treated as indicated under Snake Poisoning.

Scorpion Bites.—The larger and more venomous scorpions are to be found in the tropics. Generally in an adult the bite is followed by no more serious consequences than those of a bee or wasp. Certain genera, however, such as Buthus, contain species which cause serious symptoms, and in children and occasionally in adults, death may follow the bite of the larger varieties. The bites of the larger scorpions cause intense pain at the seat of the wound and localized swelling sometimes spreading up the limb. The constitutional symptoms are referable to the circulation and respiration; rapid and weak pulse and frequent respirations. When death occurs it is by respiratory failure.

Treatment.—The specific treatment is by means of an antiscorpion serum obtainable from several institutions. The wound should be treated with a strong solution of potassium permanganate. For the pain Duke has recommended the injection of from 5 to 10 minims of a 5 per cent solution of cocain into the tissues about the sting; for children a smaller quantity.

Centipede Bites.—Centipedes of large size may cause more serious effects in warm climates than is usual in temperate zones. Swelling and more or less intense local inflammation of the skin and subcutaneous tissues with necrosis may follow the more severe bites. Lethargy, dizziness and stupor may supervene.

Treatment.—Treatment is the same as in scorpion bite: in the milder cases applications of warm salt solution, of baking soda, or the ice-cap. The area about the sting may be bathed with diluted ammonia water.

Poisoning by Ticks, Lice and Bugs

Tick and Louse Poisoning.—The effects of the bites of ticks are local and constitutional or general, and are to be considered quite apart from the effects caused by the infectious diseases which they may transmit at the time of the bite.

During the act of biting the secretion of the salivary glands enters the tissues around the point of entrance of the parts of the tick's proboscis. Those ticks with habits like the bedbug and which attack man by preference, such as Ornithodorus talaje and moubata as well as Argas persicus, cause intense itching, severe local pain, and sometimes such general symptoms as vomiting, diarrhea, fever sweating and even delirium. Petechial hemorrhages may develop around the wound, and ulceration may follow as a result of the bite.

Besides this class of affection there is the tick paralysis which has been observed in America and Australia. The symptoms of this are dizziness, restlessness and a staggering gait. Difficulty is experienced in picking up objects from the ground. Swallowing and speaking are difficult. In the more severe cases paralysis of the arms or legs supervenes. Cases with mental confusion and irrationality have been noted. Most of the patients were children.

Complete recovery has followed the removal of the tick. A few deaths have been reported. The affection has been encountered in animals. Recovery has followed the removal of the tick but not in those cases in which the paralysis had become general. The cases in America occurred in the states of Washington, Idaho and Montana, and the ticks responsible for the condition are Dermacentor venustus and Hæmaphysalis cinnabarina.

Preventive Treatment.—It is difficult to prevent ticks from penetrating the clothing and reaching the skin, and this is particularly the case with the larvæ or "seed" ticks. On the trails and during a march it is possible to reduce the numbers very materially by the expedient of bringing the socks outside and over carefully folded-in trousers legs. After an exposure on the trails or in the bush, the body should be anointed with any neutral greasy or oily substance which will kill the ticks, and often cause them to drop off. Menthol, albolene or ointment will relieve the itching but must not be allowed to touch the delicate skin of the scrotum. Ticks should be as carefully removed as possible so as to avoid breaking off and leaving the proboscis in the skin, where it will remain for weeks to cause itching and sometimes ulceration. In tick paralysis the tick should be searched for and removed at once, not overlooking the perineum, a favorite site. No specific treatment for tick paralysis is known.

The bites of representatives of the Hemiptera include the lice and bugs, some of which, such as the cone-nosed bug, may cause a very severe bite. These, as well as the lesions caused by certain beetles (Coleoptera), more particularly the "blister beetle," are treated by means of local applications and antiseptic dressings. In the case of the "blister beetle" a vesicating fluid is excreted by the beetle in crawling over the skin. The blister which ensues should be pricked and a carbolic lotion dressing applied, as recommended by Chalmers and King.

Poisonous Insects—Flies (Myiasis), Bees, Wasps, and Ants (Lepidoptera).—The bites of ants, bees, wasps, and hornets may be treated as a class. Some of the tropical ants produce very painful bites. The venom consists chiefly if not wholly of formic acid, and the sensation experienced after a bite is acid and burning in character. In addition to the pain at the seat of the bite, general symptoms are sometimes experienced.

The venom of bees contains in addition to formic acid some more distinctly venomous substances probably allied to the venom of snakes; indeed there is some resemblance to cobra venom.

Besides the local pain and swelling, nausea, vomiting, weakness and prostration and rarely death may supervene, after circulatory and respiratory failure.

Treatment.—These bites often occur in the bush where remedies are not obtainable. Injections of potassium permanganate solution should be made into the wound immediately after the bite. Local applications of diluted ammonia water or of moistened baking soda may be made to the bite. An ice compress will prevent excessive swelling. If there are general symptoms of prostration of cyanosis, injections of strychnin or caffein should be administered.

POISONING BY CATERPILLARS, BUTTERFLIES AND MOTHS

The barbed hairs of several species of tropical lepidopterous larvæ enter the epidermis with great facility. They bear an irritating substance which sets up a peculiar dermatitis usually urticarial in character. Children are especially liable to exposure to this affection playing about certain shrubs visited by the larvæ, such as the Hibiscus plant in Brazil. The symptoms are those of intense itching, often acutely painful. The skin of the face when affected is swollen and blanched, as in urticaria.

Treatment.—Symptoms arise very acutely and there are oftentimes no evidences of the presence of caterpillar hairs. Acute itching associated with an area of urticaria should lead one to suspect an affection of this kind. The hairs should be removed by means of gauze moistened in vinegar or a solution of baking soda, and the itching allayed by the application of menthol either directly or dissolved in spirit.

POISONOUS FLIES AND FLEAS

The bites of flies and of fleas call for little comment. Dipterous flies which cause irritation by their bites are mosquitoes and fleas.

Treatment.—Lotions of ammonia water or of carbolic acid are used to relieve the itching. Great care must be exercised in the application of diluted carbolic acid not to use too strong a preparation as fatal cases of poisoning have arisen following the application of the acid to too large an area of skin. Alcohol and oil of bergamot or other essential oil may be used to relieve the itching when severe.

MYIASIS

The invasion of the body by dipterous larvæ is brought about in various ways and a number of species of flies are concerned in the infection. For the most part the larvæ are attracted to man by some pathological condition from which he suffers which gives off a peculiar pungent odor. This is an odor associated with putrefaction and is sought from long distances by the flies in question so that they may lay their eggs in locations which will yield food for the growing larvæ. The odor produced by spirochetes on squamous epithelium in cases of zena is powerfully attractive and the female screw-worm fly Cochliomyia (Chrysomyia, Compsomyia) macellaria will fly long distances in order to locate these favorable situations, and will deposit her eggs in the nostrils of a sleeping person having an offensive catarrh. The eggs hatch into larvæ which enter the nasal passages, and begin feeding on the mucous membrane. Later they enter the accessory nasal sinuses causing nosebleed and a bloody foul-smelling discharge. Often infection, septicemia and death result. Other species of flies are concerned in causing rhinal myiasis in other parts of the world, but the rationale of the infection is the same.

Aural and ocular myiasis are caused in a similar way by larvæ of Œstrus ovis, Calliphora and Sarcophaga. Cutaneous wounds of any kind are also likely to be visited by flies for the same purpose, particularly when the wounds have become infected and neglected. Urinary and vaginal infections also occur and the larvæ have crawled along the subcutaneous tissues of the penis and the hypogastrium nearly to the umbilicus. Larvæ have been passed during a painful orgasm. Intestinal myiasis is caused whenever the larvæ have been laid upon meat or other food, particularly tainted meat when not protected, and this has been used as food. Symptoms are referable to the alimentary tract, such as colic, diarrhea, loss of appetite, gastric distress, vomiting and fever of mild or severe grade may occur.

Dermal myiasis may, however, be caused in quite another way than by the deposition of eggs on a cutaneous wound. In tropical America this infection is caused by the larvæ of the fly Dermatobia cyanniventris, and it is brought about in a very remarkable way. The eggs of the fly become attached to the ventral aspect of the abdomen of a rather large species of mosquito, Janthinosoma lutzi, and when the mosquito is feeding on man the larvæ escape and pierce the skin of the person bitten by the mosquito. The larvæ then increase in size and form, a swelling which exudes a serous and sanguinolent fluid through a small opening in the center of the swelling (the warble) which is usually indurated and reddened. Dunn has recently found Dermatobia larvæ attached to a tick and this may explain the occurrence of infections in parts inaccessible to mosquitoes, such as the scrotum, a structure often attacked by ticks.

In Africa dermal myiasis is caused by a species of Cordylobia which lays its larvæ on the clothing or floor. The larvæ penetrate the skin and take up a position within an inflammatory boil-like nodule having a central opening from which the maggots may be expelled.

Treatment.—The "screw-worm" fly and other flies of this class show great diversity in their breeding habits, and they will be found wherever there is decaying vegetable matter or putrefying carcasses of dead wild or domestic animals. While nothing can be done in the jungle to reduce their numbers, it is possible to minimize the danger of infections in towns and villages by burning or deep burial of dead animals and refuse from abattoirs. All meat after slaughtering and that placed on sale should be protected by screening. All cooked food prepared for the table should be protected from flies. There was a gradual reduction in the numbers of cases of myiasis in the Canal Zone from year to year as a result of the improved sanitary condition of the municipalities, markets, villages and hygiene of the people.

The cutaneous wounds of man should be protected by suitable dressings, those of animals by applications containing substances such as cresol, fish oil, or tar which repel flies. While flies prefer to deposit their eggs on diseased surfaces, such as in the nostrils of lepers and persons suffering from ozena, etc., eggs have been found on the normal mucosa at times. It is therefore to be recommended that, whenever it is necessary to visit unsanitary areas in native villages or the jungle, one should never go to sleep either in the daytime or at night unless thoroughly protected by a mosquito bar, for these flies usually make their visits in daylight hours.

The larvæ may be removed directly or after cocainizing the nasal mucosa and plugging the nostril with cotton soaked in chloroform. This treatment is carried out two or three days until no more larvæ are removed. Sometimes large numbers of the larvæ may be present; Reeder at Ancon removed two hundred and fifty from one patient. They burrow deeply into the tissues of the mucosa and may be detected by their movements which are accelerated by the application of some irritating substance such as a little chloroform. Care should be exercised in handling larvæ from nasal passages of lepers for acid-fast bacilli resembling lepra bacilli have been found within their intestinal tracts. In involvement of the nasal sinuses and middle ear by screw-worm larvæ, generalized bacterial infection, either pneumonia or meningitis, may supervene.

When the larvæ are in other locations, they may be expelled or removed by applications of chloroform in diluted form. After the larvæ have been removed, the injured and inflamed tissues are to be treated surgically in a manner suited to the location and the character of the lesions.

Rhinal myiasis may be treated with nasal irrigations of Acidi carbolici, ½ per cent, twice daily.

In ocular myiasis and in some cases of invasion of the nasal sinuses it may be necessary to remove the larvæ by surgical means..

In intestinal myiasis some one of the efficient vermicides such as chenopodium, carbon tetrachlorid or thymol may be used.

Dermal myiasis caused by Dermatobia cyanniventris is usually acquired in the bush, as by engineers and surveyors, in removing the jungle. Persons camping in the jungle are occasionally infected. Prevention is difficult, but would seem to require the use of measures personally applied against mosquitoes and ticks. The larvæ may sometimes be expressed from their situation in the skin by pressure. When this cannot be done conveniently, incision, after cocainizing the skin, may be resorted to.

After removal of Dermatobia, or the larvæ of the African fly Cordylobia anthropophaga, the opening in the skin should be cleansed and treated antiseptically.

THE JIGGER

Dermatophilus penetrans is a small tropical flea now widely distributed but originally found in Central and South America.

The males and females parasitize pigs and man for the purpose of sucking blood. The female flea after impregnation imbeds herself in the skin of the feet, usually near the toes, but may invade the arms and other parts of the body. Her abdomen undergoes enormous enlargement as her eggs develop and an itching and sometimes a painful inflammatory small nodule surrounds the parasite in the skin. The skin becomes lighter in tint with a small black dot in the center. If not removed, ulceration and serious inflammation and deformity may result.

Treatment.—The insect should be removed as soon as possible with a pair of long-handled needles, to prevent development of the flea. When the jigger is the size of a hemp seed or of a pea, the skin may be carefully incised and the jigger or "bicho" removed with a blunt-pointed needle. The foot may be soaked in moist dressings the better to detect other jiggers invisible in the dry skin. Several sittings are necessary in heavy multiple infections. After removal of the jiggers the wounds should be dressed antiseptically.

Prophylaxis.—In Brazil and other endemic regions no one should go about unshod the shortest distance out of doors, or even on the floors of native houses. Shoes and stockings give ample protection.

ATRIPLICISM

This condition is stated to be due to poisoning by a plant belonging to the Chenopodiaceæ, and known as Atriplex littoralis (A. serrata). Other species of the same genus of plants are also stated to be poisonous. Some authors believe that a small insect found on the plant may be the cause of the effect seen in persons handling Atriplex. The disease is reported principally from China. With this affection may be mentioned the lacquer poisoning, which also occurs in China and Japan. Other inflammations of the skin are set up by the juice of different tropical species of Rhus. The species concerned in lacquer poisoning is Rhus vernicifera. Several poisonous species are natives of America.

Atriplicism is characterized by itching, edema, pain, local anesthesias, sensitiveness to heat, blisters, and sometimes ulcers. The symptoms of lacquer poisoning are similar to those set up by the juice of Rhus toxicodendron, familiar by personal experience to so many in the United States.

Remedial Measures.—Local applications of cold, opium, and of chloroform are recommended in atriplicism. Lead and opium wash, limewater, or a solution of carbolic acid are advised in case of lacquer poisoning. Purgation in the beginning of the disease has been recommended. In weak individuals the dermatitis may be a serious matter, and good nourishment, tonics, and other hygienic measures are indicated.

LATHYRISM

This affection has been described from India, North Africa, and the Mediterranean districts of Europe. It is an intoxication, essentially a spastic spinal paralysis, and is due to eating Lathyrus sativus and other related Leguminosæ. The disease can be produced experimentally in animals. The use of this food for a period of one to several months is necessary to produce paralysis. The lateral columns of the spinal cord seem to be principally affected.

Remedial Measures.—The food causing the mischief should, of course, be at once discontinued. Counterirritation over the spine, thermocautery, application of tincture of iodin, etc., are also recommended. Nourishing food and tonics and adequate clothing are indicated, and bromids have been advocated by several observers.

ARROW POISONING

A great variety of poisonous substances are used by savage races for arrow poisons. Some of the most important of these, with their antidotes, are here discussed.

Strychnos.—One of the best known and most commonly used substances is curare (also warara, urari, wurrali, woorari, etc.), active principle curarin. This is the South American Indian arrow poison obtained from different species of Strychnos (S. toxifera, S. crevauxi, A. castelniæana, S. gubleri, etc.).

Another substance with very similar action is aker lampong, and also ipoh aker, the former from S. maingayi, and the latter from S. wallichiana.

The introduction of these poisons into the circulation by arrow wounds or otherwise is followed by swelling and pain at the point of puncture, elevation of temperature, increase in the amount of urine and the presence of sugar, slowing of the heart, gradual diminishing of respiration, and complete paralysis of the voluntary muscles.

Remedial Measures.—Barbed arrows can be removed from the tissues by inserting grooved directors to cover the barbs. Ligate above the wound, which should be incised and sucked.

A solution of potassium permanganate should be injected around the wound, which should be washed out with the same solution.

Tannic acid rubbed into the wound is also recommended. Spirits of nitrous ether, 1 to 2 teaspoonsful in water, may be given. The victim should be catheterized to prevent reabsorption from the bladder. Heart stimulation with whisky or brandy is advised by many observers. Some use a hypodermic of strychnin and atropin. Artificial respiration must be carried on, if necessary, and the patient kept warm with water bottles, etc.

Strophanthus.—The juice of this plant is used as an arrow poison in Central Africa, Dahomey, the Soudan, etc., and is variously called kombe, ine, onaye, ochimbinga, etc. Some of the species used are Strophanthus hispidus (French Soudan), S. glabrus, S. lanosus (Zambezi and Southeastern Angola), etc. This last is a variety studied by me.

Another set of substances known as wabajo, ouabaio, wabayo, etc., is obtained from various species of Akokanthera (A. schimperi, A. wouabaio, A. deflersi, etc.), and resembles Strophanthus in its mode of action.

The active principle of Strophanthus is strophanthin, a glycosid with the formula of $C_{31}H_{48}O_{12}$ (Arnaud). The active principle of Akokanthera is wabain, also a glycosid, the action of which is even stronger than that of strophanthin. These are violent heart poisons and cause death by paralyzing the heart in systole. The usual symptoms are weak pulse, increased amount of urine, increased muscular rigidity, spasms, etc.

Remedial Measures.—The wound should be treated as in curare arrow poisoning, permanganate solution or tannic acid being used.

Chloral as an antagonist to the poison should be administered. The heart, circulation, and respiration should be stimulated with brandy or whisky (2 teaspoonsful every ten or fifteen minutes), aromatic spirits of ammonia, or caffein. A saline cathartic may be given. Inhalations of chloroform or ether to relax the muscles are recommended. Some place ice to the spine. Bromids and amyl nitrite have both been recommended. Artificial respiration should be practiced when the patient is *in extremis.*

Erythrophlœum.—Much has been written by travelers from time to time regarding the very weird and extraordinary custom practiced by the savage tribes of tropical Africa for the purpose of settling disputes among themselves, and usually known as the poison test. This unfortunate custom has nearly depopulated whole districts and has been responsible for more deaths in Africa than have many important diseases. The principal poison ingredient employed in this test is, in most parts of

Africa, obtained from trees belonging to the genus Erythrophlœum (E. judiciale, E. guineense).

Other plants are also used either instead of or in connection with Erythrophlœum. Some of the most important of these are Strychnos icaja, Physostigma venenosum, Tanghinia venenifera, Adenium somalense, Menabea venenata, etc. The poison test is variously known in Africa under the names of ombulungu, ombambu, wanga, mulianti, buave or muavi, mbundu or mboundou, ikazga, etc.

The poison observed by me is obtained from Erythrophlœum guineense, with which is usually mixed the inner bark of a shrub known as Securiadaca longipedunculata, and the fruit of Tephrosia vogelii. The bark of E. guineense in small doses produces vomiting and purging; if powdered and inhaled it causes sneezing. The aqueous extract, when injected into the skin of animals, sets up vomiting and is said to irritate and afterward paralyze the vagus nerve, causing the heart to slow and finally to stop. I administered a piece of the bark 2 inches square and ½ inch thick, ground up and mixed with soup, to a small dog. Its limbs were paralyzed in twenty-one minutes, and it died in an hour and two minutes.

The active principle is erythrophlein and has been carefully studied by Lewin. The active principle of Strychnos icaja is also an alkaloid, akazgin, and resembles strychnin and brucin in its action. The active principle of Physostigma venenosum is variously known as physostigmin, calabarin, eserin, etc., and its effects are opposite to those produced by strychnin.

Remedial Measures.—Emetics should, of course, be given. The heart weakness should be combated by stimulants. Permanganate of potassium is particularly indicated as an antidote to organic poisons in general, including all those mentioned in the preceding paragraph. It should be given in 3 or 4 gr.-doses dissolved in plenty of water at intervals of thirty minutes for a period of two or three hours.

If the poisoning is from Strychnos the patient should be placed in a horizontal position, the spasms controlled by inhalations of chloroform or ether, and the other well-known remedies and devices against strychnin poisoning used.

In case the poison is from Physostigma venenosum the usual procedure in cases of eserin poisoning should be employed. Atropin is the best physiological antidote. In cases of poisoning by any of these substances, if breathing becomes labored, artificial respiration should be resorted to.

Preventive Measures.—Native guides, etc., will generally warn the traveler of the poisonous character of the various herbs and plants encountered. The custom of adminstering the poisoning test should be put down by administrators and not winked at, even though, as is now often the case in Portuguese West Africa, modifications (the most common of which is probably the omission of the most dangerous ingredient, Erythrophlœum) are adopted in order that it be not invariably fatal.

TROPICAL INTOXICATIONS

Poisons Used for Murder, Suicide, the Production of Abortion.—A large variety of poisonous substances are used by natives of warm countries for the purposes just named. Among these may be named snake venom, poisonous insects, for example, Epicauta ruficeps, used in the Dutch East Indies for purposes of murder, Diamphidia simplex, employed by the bushmen of Africa as arrow poison, earth rich in the spores of tetanus bacillus, and putrid meat rich in ptomains, all used for the same purpose.

A large number of poisonous plants are also employed, of which only a few prominent species can be mentioned here. Paullinia pinnata is used in Brazil, the alkaloid known as timboin, and in the Dutch East Indies Milletia sericea. In Polynesia Barringtonia speciosa, Nerium odorum, etc., are employed in the same manner. In Africa various species of Hyoscyamus and several Euphorbiaceæ are commonly taken as medicine. As abortifacients Cajanus indicus, Plumbago zeylanica, the common nutmeg, etc., are in use in several tropical countries.

The effects of Milletia are characterized by violent pain in the head, tenesmus, and collapse. Nerium contains two glycosids, neriodorin and neriodorein, both of which are strong heart poisons. The African Hyoscyamus acts as the ordinary H. niger and contains both hyoscyamin and scopolamin. The drastic substances used as abortifacients are, as a rule, not fatal, although Plumbago (active principle plumbagin) is very severe in its effects. The effects of the nutmeg (Myristica fragrans) are depressant, with headache, amblyopia, collapse, etc.

Remedial Measures.—It is usually impossible to determine the exact nature of the substance giving rise to symptoms. Emetics or the stomach pump, when the poison has been swallowed, and ligation, excision, sucking, and cauterization when the poison has been introduced by means of a wound, are always indicated.

The most generally useful antidote is permanganate of potassium, due to the fact already pointed out that all of the known poisons employed by savage people are organic in nature. The permanganate should be given in solution in doses of 2 to 4 gr. immediately after the emetic or stomach pump has been used.

In case of wounds the solution should be injected with a hypodermic syringe around the seat of the puncture. Heart stimulants and antispasmodics are, of course, to be employed when indicated.

IRRITATIVE POISONOUS PLANTS

A large number of tropical plants possess stinging and irritative juices or principles. When these substances act locally on the skin a dermatitis of greater or less severity may supervene. Rarely the poisonous substance enters the circulation and serious or even fatal symptoms may follow.

In the Rhus family the poisonous substance is toxicodendrol, a clear watery fluid. The poisonous Rhus of Japan furnishes lacquer from which the irritative lacquer disease is derived.

Many representatives of the Euphorbiaceæ contain an irritating milky juice which causes an inflammatory skin affection. One member of this family is Hippomane mancinella, the notorious manzanilla (Spanish) or "manchineel" of the British West Indies. The poisonous substance is resident in all parts of the plant, bark, leaves and fruit, and causes a painful urticaria. Animals have been known to die from having eaten the fruit which is colored and conspicuous as well as having a sweetish taste. Children and strangers to the fruit have also suffered severely and fatally from its effects when eaten.

The nettle plant Urtica as well as Primula and some of the Orchidaceæ possess a poisonous principle contained within hairy filaments. Certain tropical species bear large amounts of the acrid secretion. There is a species in Panama which causes severe constitutional symptoms with great prostration and loss of consciousness, when a large area of the plant is accidentally grasped. Teakwood and satinwood, Goa wood and vanilla beans also cause dermatitis. The seed of the cashew fruit when bitten or the fumes from the roasted fruit cause a blistering inflammation on the lips or face.

"Ackee" poisoning or the "vomiting sickness of Jamaica" is caused by eating the unsound fruit of the Blighia sapida, one of the Sapindaceæ. It is also caused by eating parts of the plant or drinking the soup made from the fruit. The poison causes vomiting and severe general symptoms of collapse with great prostration and convulsions. It is often fatal. Apparently the vomiting is central in origin as well as peripheral. Scott found cellular necroses in the viscera more acute than in phosphorus poisoning.

Prophylaxis.—The disease may be prevented by avoiding the use of unsound "ackees," and the fruit is "unsound" when it is "unopened, picked from a decayed or injured branch, when it is forced open or has a soft spot."

Treatment.—The symptoms lead to death, for there is as yet no specific treatment known.

Onyalai is an acute disease observed in Africa by Wellman and Massey. It may be some form of poisoning as Mense suggests.

Large hemorrhagic bullæ break out on the tongue, soft palate and buccal and pharyngeal mucous membranes. It is sometimes fatal. Wellman observed 3 deaths in 14 cases in Africa. He recommends arsenic in full doses for the affection. Massey advises the use of large doses of cod-liver oil and baking soda.

Treatment of dermatitis caused by the irritative plant juices consists in cleansing the parts first with soap and water and afterwards with alcohol. Bland soothing lotions then may be applied. Poisoning by Manzanilla fruit is greatly mitigated whenever it has happened that large quantities of bread were eaten at the same time that the apples were swallowed. Bread seems to have diluted and locked up the poison, thus preventing its action on the mucous membranes of the alimentary tract. The administration of a large amount of soft bread pap with milk would seem to be a rational way of treating cases of poisoning by eating the fruit.

FISH POISONING

Poisoning has been caused by the eating of entirely fresh fish free from any taint produced by decomposition. Nevertheless symptoms of gastro-intestinal irritation have ensued. Species of the genus Clupea and Tetrodon living in Oriental waters have been studied. The poison appears to be contained in the genital organs, and symptoms are especially likely to come on after eating the roe.

The symptoms are those of abdominal distress, associated with headache, burning sensation in the fauces, nausea, abdominal pains and collapse. Death occurs from circulatory and respiratory failure.

Treatment.—The stomach should be emptied without delay, using an emetic. Apomorphin hydrochlor. $\frac{1}{10}$ to $\frac{1}{5}$ gr. may be used for an adult.

INDEX

NOTE: Therapeutic agents appear in black-face type, as **Balneology.**
Subjects other than treatment appear in plain capitals, as Acromegaly.
All headings are alphabetized and the relation of the sub-headings shown by indentation.

Abscess, kidney. *See* Kidney, diseases of.
— lung. *See* Lungs, diseases of.
— tooth, in chronic nasal sinusitis, 14
Acetaldehyd, as industrial poison, 687
Acetanilid, in insulation, 811
Acetylsalicylic acid, in pyelitis, 585
Acne. *See* Industrial diseases.
Aconite, effect on pulmonary pressure of, 91
— in bronchitis, 42
Aconite root, in acute laryngitis, 20
Acridin, as industrial poison, 687
Acriflavine, in bladder tumor, 636
Acrolein, as industrial poison, 687
Actinic rays, effect of, 801, 806
Actinomycosis. *See* Industrial diseases.
Active congestion. *See* Congestion, active.
Acupuncture, in muscular rheumatism, 488
Acute bacterial endocarditis, 219
— *See also* Heart, diseases of.
Acute bronchitis. *See* Bronchi, diseases of.
Acute coronary obstruction, 285
— *See also* Heart, diseases of.
Acute endocarditis, 219
— *See also* Heart, diseases of.
Acute fibrinous pleurisy. *See* Pleura, diseases of.
Acute myocarditis, 222
— *See also* Heart, diseases of.
Acute nephritis. *See* Kidney, diseases of.
Acute pericarditis, 187
— *See also* Heart, diseases of.
Acute syphilitic aortitis, 439.
Adenocarcinoma of bladder. *See* Bladder, diseases of.
Adenitis, bronchial. *See* Bronchi, diseases of.
Adenoids, acute bronchitis and, 34
— bronchopneumonia and, 102
— chronic laryngitis and, 23
— chronic rhinitis and, 6
— tuberculosis of mediastinal lymph nodes and, 78
Adhesive pericarditis, 204
Adrenalin in, bladder tumor, 635
— bronchopneumonia, 109
— collapse from thoracentesis, 160
— embolism, 457
Adrenalin in, hematuria, 504
— nephritis, 551
— phlebitis, 464
— pulmonary edema, 96
Adrenalin chlorid, in pleurisy, 161
African lethargy. *See* Trypanosomiasis.
Air-pressure, in caisson-disease, 696-698
Alcohol, glycerine and, in industrial infections, 659
— methyl. *See* Methyl alcohol, 681
— prohibition in lithiasis, 605
Alcoholism, chronic bronchitis and, 53
Aleppo boil. *See* Leishmaniasis, dermal.
Alkalies in, enuresis, 626
— lithiasis, 606
— nephritis, 545, 550, 554, 560
Alkaline salts, in pyelitis, 585
Alkaline solutions, antiseptic, in ozena, 19
Alkaline waters, in lithiasis, 605, 606
Alum-carbolic mouth wash in sprue, 822
Amino compounds of benzol and its homologues. *See* Anilin.
Ammonia as industrial poison, 687
Ammonia in, embolism, 457
— thrombosis, 448
Ammonium chlorid, in bronchitis, 47
Amputation in, aortic embolism, 456
— embolism of extremities, 455
Amyl nitrite in, angina pectoris, 278
— arrow poisoning, 902
— arteriosclerosis, 372
— essential hypertension, 400
— hemoglobinuria, 507
— pulmonary edema, 97
Amyloid disease of kidney. *See* Kidney, diseases of.
Anaphylactic shock, as reaction to serum treatment for bronchopneumonia, 104
Anastomosis of ureter and skin, in tuberculous cystitis, 619
Anemia, aplastic, following x-ray burns, 739
Anemia, secondary, in trichocephaliasis, 886
Anemia in, chronic bronchitis, 52
— essential hypertension, 417
— visceral leishmaniasis, 859
Aneurysm, aortic, 441
— *See also* Blood vessels, diseases of.

ANEURYSM, THORACIC, 443
— *See also* Blood vessels, diseases of.
ANGINA PECTORIS, 272
— in essential hypertension, 395
See also Heart, diseases of.
ANOREXIA, in renal tuberculosis, 614
Anthelmintics in, intestinal distomiasis, 873
— teniasis, 876
ANTHRACOSIS. *See* Industrial diseases.
ANTHRAX. *See* Industrial diseases.
Antidiphtheritic serum, in ozena, 19
ANTIMONY, as industrial poison, 663
Antimony in, bronchitis, 42
— dermal leishmaniasis, 865
— spasmodic laryngitis, 23
— trypanosomiasis, 850
— visceral leishmaniasis, 860
Antipyrin in myositis, 484
Antiseptics, urinary, in pyelitis, 584
Antisyphilitic treatment in paroxysmal hemoglobinuria, 507
Antitoxin in, epistaxis, 18
— membranous laryngitis, 24
— tuberculosis of the larynx, 27
ANTS, STINGS OF, 896
ANURESIS, in renal colic, 602
AORTIC ANEURYSM, 441
— *See also* Blood vessels, diseases of.
AORTIC EMBOLISM, 456
AORTITIS, SYPHILITIC, 439
— *See also* Blood vessels, diseases of.
Aortotomy in aortic embolism, 456
Apomorphin, in bronchitis, 42, 61
Apomorphin hydrochlor, in fish poisoning, 906
APOPLEXY in essential hypertension, 414
ARACHITIC BRONCHITIS. *See* Bronchi, diseases of.
Argochrom in endocarditis, 213
Argyrol, injection, in enuresis, 627
— irrigations, in acute cystitis, 618
Aristol, in membranous rhinitis, 8
ARROW POISONING, 901
ARSENIC, as industrial poison, 664
Arsenic in, arteriosclerosis, 373
— endocarditis, 213
— hemoglobinuria, 507
— onyalai, 906
— Oroya fever, 830
— phlebitis, 465
— syphilitic aortitis, 440
Arsphenamin in, angina pectoris, 283
— filariasis, 881
— frambesia tropica, 841
— paralysis of bladder, 621
— relapsing fever, 838
— syphilis of respiratory tract, 30
— syphilitic aortitis, 440
— thoracic aneurysm, 444
ARTERIOSCLEROSIS, 365
— in etiology of chronic myocarditis, 232
— *See also* Blood vessels, diseases of.
Arteriovenous anastomosis, in thromboangiitis obliterans, 476
ARTHROPODS, affections due to venomous, 894
Artificial respiration in, arrow poisoning, 902
— carbon monoxid poisoning, 680
— collapse from thoracentesis, 160
— electrical shock, 745
— embolism, 457
— industrial intoxications, 675, 677, 681, 682, 683, 684
— snake poisoning, 892
Ascites in cardiac failure with edema, 409
Aspiration of, blood in pulmonary edema, 97
— body cavities in nephritis, 557
Aspirin in, acute laryngitis, 20
— myositis, 484
— phlebitis, 464
— pyelitis, 585
ASTHMA, in hyperplastic ethmoiditis, 17
Astringents in, chronic laryngitis, 22
— epistaxis, 18
ATHEROSCLEROSIS. *See* Arteriosclerosis.
Atophan, in muscular rheumatism, 488
Atoxyl in, filariasis, 881
— prophylaxis of trypanosomiasis, 856
— trypanosomiasis, 850
ATRIPLICISM, 900
Atropin in, arrow poisoning, 902, 903
— collapse from thoracentesis, 160
— lead poisoning, 671
— nephritis, 550
— pulmonary edema, 96, 97
— pulmonary emphysema, 118
— renal colic, 601
Atropin sulphate, in enuresis, 631
AURAL DISTURBANCES, in caisson-disease, 693
AURICULAR FIBRILLATION, 301, 405
— *See also* Blood vessels, diseases of; Heart, diseases of.
AURICULAR FLUTTER, 299
— *See also* Heart, diseases of.
Autoserotherapy, in treatment of pleurisy, 161
AVERY METHOD of determining type of pneumonia, 103

BACILLUS COLI, in cystitis, 617
Balsam of Peru, in industrial infections, 659
Bandaging, for floating kidney, 502
Baths, carbonic acid, in pericarditis, 197
— hot in, anuresis, 503
— — renal colic, 597
— ice-cold, in insulation, 810
Baths, salt in, bronchiectasis, 61
— — tuberculosis of mediastinal lymph nodes, 179
Baths in, chronic bronchitis, 49
— irritable heart, 319
— nephritis, 528
— pyelitis, 582
BATRACHIA, 892
"Bayer 205," in trypanosomiasis, 852
BEES, STINGS OF, 896
Belladonna in, bronchitis, 44
— enuresis, 620, 626

“BENDS.” *See* Caisson-disease.
BENZIDIN, as industrial poison. *See* Anilin.
BENZIN, as industrial poison, 675
BENZOL, AMINO COMPOUNDS OF, and its homologues, 674
BENZOL, as industrial poison, 675
Benzyl-benzoate, in thrombo-angiitis obliterans, 475
Bichlorid of mercury instillation, in tuberculous cystitis, 619
Bichorlid of mercury solution, in industrial infections, 659
Biebrich red. *See* Scarlet red.
Bier suction cylinders, in thromboangiitis obliterans, 476
BISKRA BUTTON. *See* Leishmaniasis, dermal.
Black wash, in paronychia, 793
BLADDER, diseases of, 617
— — cystitis, 617
— — — acute, 618
— — — — treatment, 618
— — — — — argyrol, 618
— — — — — potassium permanganate, 618
— — — — — silver nitrate irrigations, 618
— — — Bacillus coli, 617
— — — catheter infection, 618
— — — chronic, 618
— — — — treatment, 618
— — — gonorrheal, 617
— — — — prophylaxis, 617
— — — prophylaxis, 617
— — — pyogenic infections and, 617
— — — — prophylaxis, 617
— — — tuberculous, 619
— — — — primary focus, 619
— — — — prophylaxis, 617
— — — — treatment, 619
— — — — — bichlorid of mercury instillations, 619
— — Hunner ulcer, 619
— — — treatment, 619
— — — — anastomosis of ureter and skin, 619
— — — — carbolic acid instillations, 619
— — — — cauterization, 619
— — — — gomenol oil instillations, 619
— — — — iodoform in oil instillations, 519
— — — — thallin sulphate instillations, 619
— — neuroses, 619, 622
— — — enuresis, 619, 623
— — — — causes, 619, 623
— — — — — cystitis, 627
— — — — — epilepsy, 624
— — — — — pyelitis, 627
— — — — — spina bifida occulta, 624
— — — — — urethritis, 627
— — — — — urinary and pelvic organs, diseases of, 624, 626
— — — — essential or idiopathic, 624
— — — — — causes, 624
— — — — treatment, 619, 624
BLADDER, diseases of, neuroses, enuresis, treatment, adenoids, removal of, 629
— — — — — alkalis, 626
— — — — — argyrol injection, 627
— — — — — atony of sphincter, 626
— — — — — atropin sulphate, 631
— — — — — belladonna, tincture of, 626, 631
— — — — — circumcision, 628
— — — — — colon bacilluria, 626
— — — — — constipation, relief of, 628
— — — — — diet, 625, 630
— — — — — drugs, 620, 626, 630
— — — — — electrical, 627
— — — — — general, 630
— — — — — hyoscyamus, tincture of, 626, 631
— — — — — silver nitrate, 627
— — — — — strychnin sulphate, 631
— — — — — surgical, 627
— — — — — thyroid extract, 620, 629
— — — — — urotropin, 626
— — — — — vaccine, colon, 628
— — — — — vesical sedatives, 627
— — — — urinary and pelvic organs, diseases of, 626
— — — — — treatment, 627
— — — — functional, 619
— — — paralysis of bladder, 621, 622
— — — — atony distinguished from, 621, 622
— — — — causes, 621, 622
— — — — — diphtheria, 621
— — — — — spinal lesion, 621
— — — — — tabes, 621
— — — — treatment, 621, 622
— — — — — arsphenamin, 621
— — — — — catheter, use of, 621, 623
— — — — — electricity, 623, 630
— — — — — ergot, 623
— — — — — strychnin, 623
— — — reflex, 620
— — — — causes, 620
— — — — — faradization of urethral mucosa, 620
— — — — — hygiene, 620, 630
— — — — — remote organs, diseases of, 624, 628
— — tumors, 633
— — — classification, 633
— — — of connective tissue origin, 633
— — — — fibromyxoma, 633
— — — — myxoma, 633
— — — — sarcoma, 633
— — — of epithelial origin, 633
— — — — adenocarcinoma, 633
— — — — carcinoma, basal, 633
— — — — — localized circumscribed infiltrating, 633
— — — — — squamous-celled, 633
— — — — — superficial non-infiltrating papillary, 633
— — — — cysts, 633
— — — — papilloma, 633
— — — references, 650
— — — symptoms, 633

BLADDER, diseases of, tumors, classification, constitutional and renal, 634
————cystitis, 634
————dysuria, 634
————hemorrhage, 633
————pain, 634
————retention, 634
———treatment, 636
————cystectomy, 648
————cystoscopic or urethroscoptic, 643
————cystostomy, 648
————emanation, radium, 643
————fulguration, 636
—————novocain as anesthetic, 636
—————technic, 637
————operation, 638
—————suprapubic cystotomy, 638
————radiation, 643
————radium seeds, implantation, 648
————ureteral transplantation, 648
————x-ray therapy, 648
———types of growth, treatment best suited to, 650
Blisters, in local treatment of pericarditis, 194
Blood transfusion, before operation, 453
Blood transfusion in, endocarditis, 214, 215, 221
—industrial intoxications, 675, 677, 681, 684
—methemoglobinuria, 506
—Oroya fever, 830
—thrombosis, 448
—uremia, 427
Blood urea tests, in nephritis, 521
BLOOD VESSELS, diseases of, 365
——aortic aneurysm, 441
———pressure signs, 441
————bones, 442
————circulatory system, 442
————esophagus, 442
————nervous system, 441
————respiratory system, 441
———treatment, 442
————diet, 442
————drugs, 443
————obliteration of sac, 442
————regulation of bowels, 442
————rest, 442
——arteriosclerosis, 365
———cerebral, 374
————treatment, 374
———claudication, intermittent, 376
————treatment, 376
———coronary sclerosis, 373
————treatment, 373
———definition, 365
———etiology, 365
————diphtheria, 366
————endocrin disturbances, 366
————infections, 366
————influenza, 366
————obesity, 366
————plethora, 366
————rheumatic fever, 366

BLOOD VESSELS, diseases of, arteriosclerosis, etiology, "shoddy" arteries, 366
————syphilis, 366
————typhoid fever, 366
————wear and tear, 366
———hypertensive, 377
————treatment, 377
—————venesection, 377
—————*See also* Blood vessels, essential hypertension, 378
———infections, 366, 377
————treatment, 377
———obliterating endarteritis, 375
————treatment, 375
—————alternating ice and hot foot baths, 375
—————bromids, 375
—————constricting bandages, 375
———pathology, 366
————Monckeberg's sclerosis, 367
———renal sclerosis, 374
————treatment, 374
———senile, 366, 376
————treatment, 376
———symptoms, 367
————diffuse sclerosis, 367
————essential hypertension, 367
———toxic, 377
———treatment, 368
————bowel regulation, 369
————diet, 368
————electricity, 370
————exercise, 369
————habits, 369
————hydrotherapy, 370
————massage, 370
————medicinal, 372
—————amyl nitrite, 372
—————arsenic, 373
—————calomel, 373
—————chloral, 373
—————fibrolysin, 372
—————iodids, 372
—————iron, 373
—————nitrites, 372
—————nitroglycerin, 372
—————theobromin, 372
————occupation, 371
————radium, 371
————rest, 371
————surgical, 375
————symptomatic, 373
———types, 365, 376
————treatment, 376
——embolism, 450
———aortic, 456
————treatment, 456
—————amputation, 456
—————aortotomy, 456
———arteritis and, 450
———cachetic conditions and, 451
———cardiac, 456
———causes, 450
———cerebral, 457
————treatment, 457
———fat, 450, 454
————treatment, 454

Blood vessels, diseases of, embolism, fat, treatment, Esmarch bandage, 454
——— of coronary arteries, 456
———— treatment, 457
————— morphin, 457
————— rest, 457
————— *See also* Heart, diseases of.
——— of extremities, 455
———— treatment, 455
————— amputation, 455
————— embolectomy, 455
————— morphin, 455
——— of mesenteric vessels, 458
——— phlebitis and, 450
——— pulmonary, 457
———— treatment, 457
————— artificial respiration, 457
————— drugs, 457
——— renal, 458
——— splenic, 458
——— surgical operations and, 450
——— symptoms, 451
——— thrombosis and, 450
——— treatment, 451
———— general, 451
———— preventive, 451
————— after surgical operations, 451
——— Zahn, emboli of, 456
—— essential hypertension, 378
——— arteriosclerosis and, 378
——— cardiac involvement, 391
———— angina pectoris, 395
————— abdominal, 398
————— acute, 399
————— causes, 396
————— fatigue, 396
————— menopause and, 396
————— prognosis, 396
————— syphilis, 396
————— toxic factors, 397
————— treatment, 398
———— auricular fibrillation, 405
————— treatment, 406
———— cardiac failures with edema, 407
————— treatment, 408
———— cardiac murmurs, 410
———— coronary thrombosis and embolism, 403
————— treatment, 404
———— dyspneas of acute onset, 392
————— cardiac asthma, 392
————— dyspnea of acidosis, 393
————— paroxysmal dyspnea, 393
————— treatment, 392
————— uremic dyspnea, 393
———— early cases of cardiac inefficiency, 391
————— symptoms, 391
————— treatment, 392
———— high blood-pressure statis, 404
————— treatment of, 404
———— infarcts, pulmonary, 395
————— treatment, 395
———— paroxysmal tachycardia, 410
————— treatment, 411

Blood vessels, diseases of, essential hypertension, cardiac involvement, premature systoles, 412
————— treatment, 412
———— pulmonary edema, 394
————— treatment, 394
——— cases showing symptoms, 385
———— treatment, 385
——— cases with high diastolic pressure, 385
——— cases with low diastolic pressure, 384
——— cases without symptoms, 385
———— treatment, 385
——— cause unknown, 379
——— dietic factors in, 380
——— early cases, 381
———— prognosis, 381
———— treatment, 382
————— diet, 382
————— rest, 382
————— sedatives, 382
——— etiology, 379
——— familial disease, 380
——— later cases, 382
———— blood-pressure readings, 382
———— treatment, 385
————— rest, 385
——— myomata uteri and, 417
——— nephritis and, 378
——— renal lesions, 418
———— diagnosis, 419
————— acidosis determination, 422
————— blood chemistry, 419
————— concentration test, 419
————— creatinin, 421
————— phenolsulphonephthalein test, 421
————— salt, 421
————— sugar, 421
————— urea nitrogen, 420
————— uric acid, 420
———— treatment, 422
————— diet, 422
————— digitalis, 423
————— rest, 423
———— uremia, 423
————— convulsions, 428
————— headaches, 428
————— pathology, 423
————— suppression of urine, 428
————— symptoms, 424
————— treatment, 425
——— terminal infection, 429
——— treatment, 382
———— bathing, 387
———— clothing, 386
———— diet, 387
————— fats, 390
————— fluids, 390
————— proteins, 387
————— quantity, 387
————— salt, 387
————— sugar, 387
———— exercise, 387
———— habits of life, 386
———— summary, 430-438
——— types commonly subject to, 380

Blood vessels, diseases of, essential hypertension, vascular accidents, 412
———— anemia, 417
————— Bright's disease confused with, 419
———— cerebral vessels, 412
————— symptoms, 413
————— treatment, 413
———— erythrocytosis, 417
————— Geisbock's disease and, 417
———— hemorrhages, 416
————— epistaxis, 416
————— lungs, 417
————— meningeal, 417
————— stomach, 417
————— urinary tract, 417
————— uterus, 417
———— *See also* Vascular accidents, cerebral vessels; retinal vessels.
———— retinal vessels, 415
———— thrombosis, 415
————— mesenteric, 415
————— peripheral vessels, 416
————— symptoms, 415, 416
————— treatment, 415, 416
—— phlebitis, 458
——— chronic, 462
———— diagnosis, early, 463
———— general arteriosclerosis and, 462
———— symptoms, 462
——— frequency, 458
——— mechanical activity and, 460
——— non-obliterative, 461
———— symptoms, 461
——— obliterative, 461
———— symptoms, 461
——— recurrent, 462
———— surgical care in, 462
———— symptoms, 462
——— references, 468
——— syphilis and, 459
——— thrombosis and, 459
——— treatment, 463
———— bandaging, 466
———— diet, 464
————— gouty or nephritic cases, 464
———— drugs, 464
———— electricity, 467
———— fomentations, warm to hot, 466
———— heat, 464
———— hydromineral, 467
———— immobilization, 463
———— lavage, colonic, 464
———— sunlight, 467
———— surgical, 467
————— removal of vein, 467
————— resection of vein, 467
———— water, 465
——— typhoid fever and, 459, 460
——— varicose ulcers and, 460
—— syphilitic aortitis, 439
——— acute, 440
———— treatment, 440
————— arsenic, 440
————— arsphenamin, 440

Blood vessels, diseases of, syphilitic aortitis, treatment, diet, 440
————— mercury, 440
————— potassium iodid, 440
————— rest, 440
————— symptomatic, 440
——— chronic, 441
———— treatment, 441
——— clinical features, 439
——— treatment, 439
—— thoracic aneurysm, 443
——— treatment, 443
———— bowel regulation, 443
———— diet, 443
———— drugs, 443
————— digitalis, 445
————— sedatives, 444
————— vasodilators, 444
———— management after, 445
———— rest, 443
———— surgical, 444
————— Macewen's operation, 445
————— Matas' method, 445
————— wiring and electrolysis, 444
———— symptomatic, 444
————— morphin, 444
————— venesection, 444
—— thrombo-angiitis obliterans, 469
——— age and, 471
——— definition, 469
——— diagnosis, 472
——— etiology, 469
———— alcoholic habits, 469
———— exposure to cold, 470
———— syphilis, 469
———— tobacco, 469
———— unstable nervous system, 469
——— history and geographical distribution, 470
——— pathology, 472
——— prognosis, 474
——— race and, 471
——— sex and, 471
——— symptoms, 472
——— treatment, 475
———— arteriovenous anastomosis, 476
———— Bier suction cylinders, 476
———— drugs, 475
———— electrotherapy, 476
———— elevation of member, 475
———— femoral vein ligation, 476
———— heat, 475
———— injections, intravenous and subcutaneous, 477
———— Leriche's periarterial sympathectomy, 478
—— thrombosis, 446
——— cardiac, 449
———— auricular dilatation and, 449
———— cause, 449
———— diagnosis, 449
———— treatment, 449
————— drugs, 449
——— infectious diseases and, 446
——— lymphangitis and, 446
——— marantic conditions and, 446
——— mesenteric, 450

BLOOD VESSELS, diseases of, thrombosis, mesenteric, treatment, 450
— — — pathology, 446
— — — peripheral, 450
— — — — *See also* Phlebitis.
— — — pneumonia and, 446
— — — pulmonary, 449
— — — — marantic conditions and, 449
— — — — pneumonia and, 449
— — — — typhoid fever and, 449
— — — — treatment, 449
— — — — — drugs, 449
— — — — — oxygen inhalations, 449
— — — — — rest, 449
— — — surgical conditions and, 446
— — — treatment, 447
— — — — drugs, 448
— — — — electric ionization of potassium iodid, 448
— — — — ligature, 448
— — — — prophylactic, 447
— — — — transfusion, 448
— — — typhoid fever and, 446
— — *See also* Heart, diseases of.
BLOOD-PRESSURE READINGS, precautions to be taken in, 382
BOBAS. *See* Tropical diseases, frambesia tropica.
Boghean apparatus in pulmonary emphysema, 117
BONES, diseases of, 492
— — osteitis deformans, 492
— — — definition, 492
— — — etiology, 492
— — — pathology, 492
— — — references, 496, 497
— — — symptoms, 492
— — — treatment, 493
— — — — diet, 493
— — osteo-arthropathy, secondary hypertrophic, 495
— — — — definition, 493
— — — — etiology, 493
— — — — pathology, 494
— — — — references, 497
— — — — symptoms, 494
— — — — treatment, 494
— — osteomalacia, 494
— — — clinical forms, 495
— — — course and progress, 495
— — — definition, 494
— — — etiology, 494
— — — — glands of internal secretion, 494
— — — pathology, 495
— — — references, 497
— — — symptoms, 495
— — — treatment, 495
— — — — castration, 495
— — — — cod-liver oil, 496
— — — — diet, 495
— — — — drugs, 496
— — — — gland therapy, 496
— — — — hot packs, 496
— — — — rest, 495
Boric acid solution, saturated, in industrial infections, 659
BOWMAN'S THEORY of renal secretion, 509
BRASS, as an industrial poison, 665
BRASS FOUNDER'S AGUE, 666
Brauer's air pressure apparatus, for lung tumors, 127
Bromids in, arrow poisoning, 902
— arteriosclerosis, 375
— insulation, 811
— phlebitis, 464
BROMIN, as industrial poison, 687
Bromur, in bronchitis, 44
BRONCHI, diseases of, 32
— — adenitis, 67
— — — complicating bronchopneumonia and bronchitis, 67
— — — influenza accompanied by, 67
— — — measles accompanied by, 67
— — — treatment, 67
— — — — drugs, 68
— — — — sea air, 68
— — — — tuberculin, 68
— — — — x-ray, 68
— — — tuberculosis of children accompanied by, 67
— — — whooping-cough accompanied by, 67
— — bronchiectasis, 61
— — — treatment, 61
— — — — baths, salt, 62
— — — — climate, 62
— — — — diet, 62
— — — — environment, 61
— — — — posture, 62
— — — — surgical, 63
— — — — symptomatic, 64
— — — — — aromatic inhalations, 65
— — — — — cough, 64
— — — — — creosote, 64
— — — — — expectorants, 65
— — — — — fetor, 64
— — — — — injections, intratracheal, 66
— — — — — — nitrogen gas, 64
— — — — — oxygen inhalation, 65
— — — — — Trendelenburg position, 63
— — bronchitis, 32
— — — acute, 32
— — — — adenoids and, 34
— — — — cyanosis, 45
— — — — dyspnea, 35
— — — — emphysema complicated by, 35
— — — — etiology, 32
— — — — nasal polypi and, 34
— — — — tonsils, enlarged, and, 34
— — — — treatment, 32
— — — — — aconite, 42
— — — — — ammonium chlorid, 47
— — — — — antimony, 42
— — — — — apomorphin, 42
— — — — — baths, 33
— — — — — belladonna, 44
— — — — — bronchitis kettle, 35
— — — — — camphor, 36
— — — — — climate, 33
— — — — — codein, 43
— — — — — cold applications, 33
— — — — — cupping, 45
— — — — — diet, 36

Bronchi, diseases of, bronchitis, acute, treatment, emetics, 46
— — — — — expectorants, 41
— — — — — heroin hydrochlorid, 43
— — — — — laxatives, 41
— — — — — leeches, 39
— — — — — liniments, stimulating, 38
— — — — — lobelia, 44
— — — — — local, 38
— — — — — opium, 42
— — — — — oxygen, inhalation of, 45
— — — — — potassium bichromate, 47
— — — — — potassium iodid, 47
— — — — — poultices, 38
— — — — — quinin, 36
— — — — — soporifics, 43
— — — — — sprays, 40
— — — — — stimulating expectorants, 44
— — — — — strychnin, 45
— — — — — symptomatic, 37
— — — — — temperature of room, 34
— — — — — turpentine, 58
— — — — — vapors, 39
— — — chronic, 47
— — — — alcoholism and, 53
— — — — anemia in, 52
— — — — children and, 52
— — — — contagion, 50
— — — — cough, 55
— — — — cyanosis, 60
— — — — dyspnea, 60
— — — — emphysema, 60
— — — — gout and, 53
— — — — insomnia, 55
— — — — — treatment, 55
— — — — obesity and, 52
— — — — profuse expectoration, 57
— — — — tobacco and, 50
— — — — treatment, 48
— — — — — baths, incandescent light, 60
— — — — — — salt, 49
— — — — — climate, 48
— — — — — clothing, 50
— — — — — cod-liver oil, 52
— — — — — diet, 51
— — — — — drugs, 54
— — — — — dust, avoidance of, 50
— — — — — environment, 48
— — — — — exercises, 51
— — — — — expectorants, 54, 58
— — — — — liniments, 55
— — — — — mouth breathing, avoidance of, 49
— — — — — poultices, 55
— — — — — serum, 53
— — — — — sprays, 55, 56, 59
— — — — — strychnin, 55, 60
— — — — — symptomatic, 54
— — — — — temperature, 50
— — — — — vaccine therapy, 53
— — — — — whey cure, 52
— — — — — x-ray, 59
— — — — uremia, 53
— — — plastic, 60
— — — — nature, 60
— — — — treatment, 60
— — — — — apomorphin, 61
Bronchi, diseases of, bronchitis, plastic, treatment, emetics, 61
— — — — — iodid of potassium, 60
— — — — — ipecacuanha, 61
— — — — — pilocarpin, 61
— — foreign bodies in bronchi, 72
— — — air passages, 77
— — — — diagnosis, 81
— — — — — inspection, 84
— — — — — palpation, 84
— — — — — percussion, 85
— — — — — physical signs, 81
— — — — symptoms, 77
— — — — — bronchial foreign body, 80
— — — — — esophageal foreign body, 84
— — — — — initial, 78
— — — — — laryngeal foreign body, 78
— — — — — tracheal foreign body, 79
— — — — treatment, 85
— — — — — after bechic expulsion of body, 86
— — — — — bronchoscopic removal, 86
— — — — — coughing up foreign bodies, 85
— — — — — disintegration, 86
— — — — — thoracotomy, 86
— — — — — tracheotomy, 86
— — — arachidic bronchitis, 74
— — — — pathological changes, 74
— — — lung-suppuration, 75
— — — — atelectate area in, 76
— — — — drowned lung, 76
— — — — foreign body causing, 75
— — — — pulmonary abscess, 76
— — — pathology, 72
— — — — metallic bodies, 72
— — — — vegetable substances, 73
— — — references, 88
— — — Roentgenogram showing, brass paper fastener in lung, 75
— — — — lung in which safety pin remained two months, 79
— — — — lung seven years after removal of paper fastener, 76
— — — — lung with shawl pin in left lobe, 80
— — — — peanut kernel in right main bronchus, 82
— — — — pulmonary abscess due to screw, 77
— — — — safety pin in lung, 74
— — — — shawl pin in lung, 73
— — — — small electric lamp in lung, 82
— — references, 69-71
Bronchial adenitis. *See* Bronchi, diseases of.
Bronchiectasis. *See* Bronchi, diseases of.
Bronchitis, arachidic. *See* Bronchi, diseases of.
Bronchitis. *See* Bronchi, diseases of.
Bronchitis, acute. *See* Bronchi, diseases of.
Bronchitis, chronic. *See* Bronchi, diseases of.
Bronchitis, plastic. *See* Bronchi, diseases of.

BRONCHOPNEUMONIA. *See* Lungs, diseases of.
BRUCELLA MELITENSIS, causing undulant fever, 812
BRUCELLA PARAMELITENSIS, causing undulant fever, 812
BUBAS. *See* Tropical diseases, frambesia tropica.
BUBOES, CLIMATIC. *See* Tropical diseases, local inflammations.
BUGS, poisoning by, 896
BURNS, ELECTRICAL, 742
— radium, 787
— x-ray, 787
— *See also* Industrial diseases.
BUTTERFLIES, poisoning by, 897

CADMIUM, as industrial poison, 689
Caffein in, congestion of lungs, 94
— methemoglobinuria, 506
— nephritis, 545, 549, 551, 554, 560
—nitrobenzol poisoning, 682
— pleurisy, 148
Caffein sodium, in treatment of bronchopneumonia, 109
CAISSON DISEASE. *See* Industrial diseases.
CALABAR SWELLINGS. *See* Filaria loa.
Calsium carbonate, in sprue, 822
Calcium lactate in, hematuria, 504
— sprue, 822
CALCULUS, RENAL, as a cause of hydronephrosis, 592
— *See also* Kidney, diseases of, nephrolithiasis.
CALLOSITAS. *See* Industrial diseases.
CALLUS. *See* Callositas.
Calomel in, acute laryngitis, 20
— acute bronchitis, 36
— arteriosclerosis, 373
— dengue fever, 817
— edematous laryngitis, 23
— membranous rhinitis, 8
— nephritis, 529, 553
— pleurisy, 145
Camphor in, anilin poisoning, 675
— congestion of lungs, 94
— methemoglobinuria, 506
— nitrobenzol poisoning, 682
Carbolic acid instillations, in tuberculous cystitis, 619
Carbon-arc lamp, 723
CARBON BISULPHID, as industrial poison, 677
CARBON MONOXID, as industrial poison, 679
CARBON TETRACHLORIN, as industrial poison, 680
Carbonic acid, used rectally in pulmonary edema, 97
CARCINOMA, basal-celled, of bladder. *See* Bladder, diseases of.
— localized circumscribed infiltrating of bladder. *See* Bladder, diseases of.
— of the pleura. *See* Pleura, diseases of.
— squamous-celled, of bladder. *See* Bladder, diseases of.
CARCINOMA, superficial non-infiltrating papillary, bladder. *See* Bladder, diseases of.
CARDIAC ASTHMA, 392
CARDIAC DISEASE. *See* Heart, diseases of.
CARDIAC FAILURES WITH EDEMA, 407
CARDIAC INVOLVEMENT, in essential hypertension, 391
— *See also* Blood vessels, diseases of.
CARDIAC NEUROSES, 312
— *See also* Heart, diseases of.
CARDIAC PAIN, 272.
— *See also* Heart, diseases of.
Cardiac stimulants in, treatment of myocarditis, 225
— renal colic, 602
Cardiac stimulation, in electrical shock, 744
CARDIAC THROMBOSIS, 449
Cardiolysis, in indurative mediastinopericarditis, 181
Cardiovascular stimulants, in pleurisy, 148
Castration, in osteomalacia, 495
CATARACT, GLASS WORKERS', 718
CATARRH, acute laryngeal. *See* Larynx, diseases of.
— acute nasal, acute nasal sinusitis complicating, 11
— — of respiratory tract. *See* Bronchi, diseases of.
CATERPILLARS, poisoning by, 897
Cathartics, saline, in kidney of pregnancy, 578
Cathartics in, nephritis, 529, 542, 552, 553, 560, 534
— pleurisy, 147
— suppression of urine, 603
— uremia, 426
Catheterism, in bladder tumor, 635
—ureteral, in renal calculus, 606
Catheterization of lung, in pulmonary edema, 97
Cauteries, in epistaxis, 18
Cauterization with, acid nitrate of mercury in tuberculous cystitis, 619
— high frequency current in tuberculous cystitis, 619
Caustics, in chronic laryngitis, 22
CENTIPEDE POISONING, 895
CEREBRAL EMBOLISM. *See* Embolism.
Cesarean secretion, in kidney of pregnancy, 578
CESTODE INFECTION. *See* Tropical diseases.
CHAGAS' DISEASE. *See* Tropical diseases, trypanosomiasis.
CHIGGER. *See* Jigger.
CHIGO. *See* Jigger.
CHIGRE. *See* Jigger.
CHILBLAINS, 715
Chloral in, arrow poisoning, 902
— essential hypertension, 401
— nephritis, 549
— phlebitis, 464
— renal colic, 601

Chloral hydrate in, treatment of bronchitis, 43
— kidney of pregnancy, 578
Chloramid, in treatment of bronchitis, 43
Chlorid of iron, in epistaxis, 18
CHLORIN, as industrial poison, 687
Chloroform, effect on pulmonary pressure, 91
Chloroform in, treatment of angina pectoris, 279
— arrow poison, 902, 903
— essential hypertension, 401
— kidney of pregnancy, 578
—renal colic, 600, 601
—uremia, 428
Cholecystotomy, in distomiasis, 875
CHORDITIS NODOSA, and chronic laryngitis, 22
CHOREA, in etiology of chronic myocarditis, 232
Chromic acid, in chronic rhinitis, 7
CHROMIUM COMPOUNDS, as industrial poisons, 687
CHRONIC ATROPHIC LARYNGITIS. *See* Larynx, diseases of.
CHRONIC BRONCHITIS. *See* Bronchitis, chronic.
CHRONIC INTERSTITIAL NEPHRITIS. *See* Kidney, diseases of.
CHRONIC PARENCHYMATOUS NEPHRITIS. *See* Kidney, diseases of.
CHRONIC PHLEBITIS, 463
— *See also* Blood vessels, diseases of.
CHRONIC MYOCARDITIS, 227
— *See also* Heart, diseases of.
CHRONIC PNEUMONIA. *See* Pneumonia, chronic.
CHRONIC VALVULAR DISEASE OF HEART. *See* Valvular disease of heart, chronic.
Churchill's tincture of iodin, in industrial infections, 659
Circumcision, in enuresis, 628
Citric acid in, embolism, 457
— phlebitis, 465
— thrombosis, 448, 449
CLAUDICATION, INTERMITTENT, in arteriosclerosis, 376
— — *See also* Thrombo-angiitis obliterans.
Climate in, atrophic rhinitis, 8
— bronchiectasis, 62
— chronic bronchitis, 48
— nephritis, 523, 526
— pulmonary emphysema, 116
— tuberculosis of the larynx, 26
CLIMATIC BUBOES. *See* Tropical diseases.
Clothing, in nephritis, 523
CLYLOTHORAX. *See* Pleura, diseases of.
Coagulen, in bladder tumor, 635
Coal-tars, in pyelitis, 584
COCHIN CHINA DIARRHEA, 885
Codein in, aortic or thoracic aneurysm, 443
— bronchitis, 43
— edematous laryngitis, 23
— phlebitis, 464
— thoracic aneurysm, 444
Cod-liver oil and baking soda, in onyalai, 906
Cod-liver oil in, chronic bronchitis, 52
— osteomalacia, 496
Cold applications in pericarditis, 194, 196
Cold baths, in pleurisy, 147
COLD, occupational diseases and. *See* Industrial diseases.
COLIC, RENAL. *See* Kidney, diseases of, nephrolithiasis.
COLITIS, ULCERATIVE, 866, 867
Collargol, in endocarditis, 213
Colloidal metals, in endocarditis, 213
Colloidal therapy, in undulant fever, 813
Colonic irrigation, in suppression of urine, 603
COMPRESSED AIR ILLNESS. *See* Caisson-disease.
Compresses, cold, in acute laryngitis, 20
CONGENITAL CARDIAC DISEASE, 322.
— *See also* Heart, diseases of.
CONGENITAL CYANOSIS, 325
CONGESTION OF LUNGS, active. *See* Lungs, diseases of.
— hypostatic. *See* Lungs, diseases of.
— passive. *See* Lungs, diseases of.
CONJUNCTIVITIS, ultraviolet rays causing, 724
Coolidge tube, in production of x-rays, 734
COPPER, as industrial poison, 689
CORONARY ARTERIES, embolism of, 456
CORONARY EMBOLISM, 403
— *See also* Blood vessels, diseases of.
CORONARY THROMBOSIS, 403
— *See also* Blood vessels, diseases of.
CORYZA, ACUTE. *See* Acute rhinitis.
COURBATURES. *See* Caisson-disease.
CRAMPS, MUSCULAR. *See* Muscles, diseases of.
Creolin, in ozena, 19
Creosote baths in bronchiectasis, 64
Creosote in, bronchiectasis, 64
— bronchopneumonia, 105
Cresol, in industrial infections, 659
CROOKES' TUBE, in production of x-ray, 733
CROUP, FALSE. *See* Laryngitis, spasmodic.
CROUP, MEMBRANOUS. *See* Laryngitis, membranous.
CROUP, SPASMODIC. *See* Laryngitis, spasmodic.
Croup kettle, in spasmodic laryngitis, 24
Croup tent in, acute laryngitis, 20
— membranous laryngitis, 25
CULEX FATIGOUS, as cause of dengue fever, 816
Cupping in, bronchitis, 45
— essential hypertension, 395
CUSHING'S THEORY OF RENAL SECRETION, 509
CUTANEOUS TUBERCULOSIS. *See* Tuberculosis.
CYANIDS, as industrial poisons, 687
CYANOGEN and its compounds, as industrial poisons, 687
CYSTITIS. *See* Bladder, diseases of.

Cystoscopic method of radiation, in bladder tumor, 643
Cystectomy in, bladder tumor, 648
Cystoscopy in, diagnosis of hydronephrosis, 594
— renal tuberculosis, 610
Cystotomy, suprapubic, in bladder tumor, 638
CYSTS of bladder. *See* Bladder, diseases of.

Dakin's solution in, pus infections, 794
— ulcer, 796
D'Arsonal current in, bladder tumor, 636
— tuberculous cystitis, 619
Decapsulation in, anuresis, 504
— nephritis, 556
Decompression, in prevention of caisson-disease, 701
DECRESCENT ARTERIOSCLEROSIS, 376
DELHI SORE. *See* Tropical diseases.
DEMOID CYSTS. *See* Mediastinum, diseases of.
DENGUE FEVER. *See* Tropical diseases.
Deny's filtrate in tuberculosis of larynx, 28
DERMAL LEISHMANIASIS. *See* Leishmaniasis.
DERMATITIS, acute. *See* Industrial diseases.
— radium, 737
— x-ray, 738
DERMATITIS FACTITIA. *See* Industrial diseases.
DERMATOMYOSITIS. *See* Polymyositis, acute.
DERMATOSES, OCCUPATIONAL. *See* Industrial diseases.
Desiccation, in bladder tumor, 636
Diaphoresis in, suppression of urine, 603
— uremia, 426
Diaphoretics, in pleurisy, 148
DIARRHEA in, intestinal schistosomiasis, 879
— visceral leishmaniasis, 862
Dichloramin-T, in prevention of industrial infections, 658
Diet, after thoracentesis, 162
Diet in, accidents to cerebral vessels, 413, 423
— acute pericarditis, 192
— aortic or thoracic aneurysm, 442, 443
— arteriosclerosis, 369
— bronchopneumonia, 105
— chronic bronchitis, 51
— essential hypertension, 382, 387, 402, 408, 423
— irritable heart, 319
— kidney of pregnancy, 577
— lithiasis, 604, 605
— movable kidney, 502
— muscular rheumatism, 488
— myocarditis, 224, 256, 259
— nephritis, 523, 547, 552, 554, 560, 562
— Oroya fever, 830
— osteitis deformans, 493
— osteomalacia, 495
Diet in, phlebitis, 464
— pulmonary edema, 97
— pyelitis, 582
— renal tuberculosis, 612
— sprue, 822
— syphilitic aortitis, 440
DIETHYLSULPHATE, as industrial poison, 687
Differential air pressure, in lung tumors, 126
Digifolin, in nephritis, 551, 554
DIGITALIS, effect on pulmonary pressure of, 91
Digitalis in, amyloid disease of kidney, 577
— angina pectoris, 282, 287
— anilin poisoning, 675
— auricular fibrillation, 302
— bronchopneumonia, 109
— congestion of lungs, 94
— disorders of heart beat, 298, 300, 302, 308, 311
— embolism, 457
— essential hypertension with cardiac involvement, 392, 393, 395, 399, 403, 404, 405, 406, 408, 411, 412, 423, 427
— myocarditis, 224, 236, 253, 258, 262, 264, 265
— nephritis, 546, 549, 550, 558, 560
— passive congestion of lungs, 94
— pericarditis, 196
— pleurisy, 148
— pulmonary edema, 97
— pulmonary emphysema, 114, 118
— thoracic aneurysm, 445
— thrombosis, 449
DIMETHYLSULPHATE, as industrial poison, 687
DINITROBENZOL. *See* Nitrobenzol.
DISEASES OF BONES. *See* Bones, diseases of.
DISORDERS OF HEART BEAT, 293
— *See also* Heart, diseases of.
Dispargen, in endocarditis, 213
DISTOMIASIS. *See* Tropical diseases.
Diuretics in, anuresis, 503
— nephritis, 545, 549, 553, 556
— pleurisy, 148
— pyelitis, 584
— suppression of urine, 603
— uremia, 426
DIVER'S PALSY. *See* Caisson-disease.
Dobell's solution, in laryngitis, 21
Douches, hot, in renal colic, 598
Dover's powder in, acute laryngitis, 20
— edematous laryngitis, 23
DRACUNCULUS MEDINENSIS. *See* Tropical diseases, filariasis, 882
Drainage of legs, in nephritis, 557
Drug therapy, in renal colic, 600, 601, 603
DUMDUM FEVER. *See* Leishmaniasis, visceral.
Dust, causing pneumocomosis, 772, 773
DYSENTERY, in intestinal schistosomiasis, 879
DYSPHAGIA and pericarditis, 189

DYSPNEA in, caisson-disease, 694-696
— silicosis, 773
DYSPNEA OF ACIDOSIS, 393

ECHINOCOCCUS CYSTS. *See* Pleura, diseases of, tumors.
ECLIPSE BLINDNESS, 725
ECZEMA. *See* Industrial diseases.
Edebohls' operation, in anuresis, 503
EDEMA, PULMONARY. *See* Lungs, diseases of.
— — in essential hypertension, 394
EDEMATOUS LARYNGITIS. *See* Larynx, diseases of.
EFFORT SYNDROME, 313
Ehrlich-Hatta, in filariasis, 881
ELECTRIC OPHTHALMIA, 720, 747
ELECTRICAL BURNS, 742
ELECTRICAL SHOCK, 743
ELECTRICITY. *See* Industrial diseases.
Electrocoagulation, in bladder tumor, 636
Electrotherapy in, arteriosclerosis, 370, 375
— atrophic rhinitis, 9
— enuresis, 627
— lead poisoning, 671
— movable kidney, 503
— muscular rheumatism, 488
— myositis, 484
— paralysis of bladder, 623
— phlebitis, 467
— pulmonary edema, 97
— thrombo-angiitis obliterans, 476
Elimination, through bowel and skin, in nephritis, 547, 551, 552, 560
Embolectomy, in embolism of extremities, 455
EMBOLISM, 450
— aortic, 456
— of extremities, 455
— pulmonary. *See* Lungs, diseases of.
— *See also* Blood vessels, diseases of.
Emetics, in bronchitis, 46
Emetin, in sprue, 822
EMPHYSEMA, complicated by acute bronchitis, 35
— pleurisy and, 143
— pulmonary. *See* Lungs, diseases of.
Ems water, in bronchitis, 55
ENCHONDROMA OF PLEURA. *See* Pleura, diseases of.
ENDARTERITIS, OBLITERATING, 375
ENDARTERITIS-OBLITERANS. *See* Thrombo-angiitis obliterans.
ENDEMIC HEMATURIA. *See* Tropical diseases, endemic hematuria.
ENDOCARDITIS, 207
— *See also* Heart, diseases of.
ENDOTHELIAL CARCINOMA OF PLEURA. *See* Pleura, diseases of.
Enemata, in intestinal schistosomiasis, 879
Enteroclysis, in renal colic, 599
ENTEROPTOSIS. *See* Kidney, diseases of.
EPINEPHRIN, effect on pulmonary pressure, 90
EPISTAXIS in, essential hypertension, 417
— yellow fever, 843
— *See also* Nose, diseases of.
EPITHELIOMA, 791
Ergograph, 752
Ergot in, hematuria, 504
— paralysis of bladder, 623
Ergotoxin, effect on pulmonary pressure, 90
ERYTHEMA, ultraviolet rays causing, 722
Erythrol tetranitrate in, nephritis, 543
— thoracic aneurysm, 444
ERYTHROPHLŒUM. *See* Poisons—reptiles—fish—arthropods and plants, affections due to.
Esmarch bandage, in embolism, 454
ESOPHAGEAL FOREIGN BODY. *See* Bronchi, diseases of.
ESSENTIAL HYPERTENSION, 378
— *See also* Blood vessels, diseases of.
Ether in, arrow poisoning, 902, 903
— methemoglobinuria, 506
ETHMOIDITIS, HYPERPLASTIC. *See* Nose, diseases of.
Eucalyptus, in intestinal distomiasis, 876
Exercise, moderate, after thoracentesis, 162
Exercise in, arteriosclerosis, 369
— chronic bronchitis, 51
— essential hypertension, 387, 403
— irritable heart, 318
— lithiasis, 604
Expectorants in, bronchiectasis, 65
— of bronchitis, 41, 44, 54, 58
EXTRAHAZARDOUS INDUSTRIES. *See* Industrial diseases.
EXTRASYSTOLES, 295
EYESTRAIN, 729

Factory Department of British Home Office instructions, for use of first-aid kit, 658
Faradization, in enuresis, 620
FAT EMBOLISM, 450, 454
FATIGUE, INDUSTRIAL. *See* Industrial diseases.
Fel bovis, in lead poisoning, 672
Femoral vein ligation, in thrombo-angiitis obliterans, 476
FIBRILLATION, AURICULAR, 301, 405
— — *See also* Blood vessels, diseases of; Heart, diseases of.
FIBRINOUS PLEURISY. *See* Pleura, diseases of.
FIBRONA OF THE PLEURA. *See* Pleura, diseases of.
Fibrolysin in, arteriosclerosis, 372
— thrombo-angiitis obliterans, 475
FIBROMYXOMA OF BLADDER. *See* Bladder, diseases of.
FILARIA. *See* Filariasis.
FILARIA LOA. *See* Tropical diseases, filariasis.
FILARIA PERSTANS. *See* Tropical diseases, filariasis.
FILARIASIS. *See* Tropical diseases.
First-aid kit, for industrial infections, 657

FISH, AFFECTIONS DUE TO POISONOUS. *See* Poisons—reptiles—fish—arthropods and plants, affections due to.
FISH, in destruction of Stegomyia calopus, 846
FLAGELLATE INFECTIONS. *See* Tropical diseases.
FLEAS, POISONOUS, 897
FLIES, BITES OF, 896
Flint's air pressure apparatus, in lung tumor, 127
Fluids, restriction of, in nephritis, 530, 560
FLUKES. *See* Tropical diseases, distomiasis.
FLUORIC ACID, as an industrial poison, 687
FLUTTER, AURICULAR, 299
— — *See also* Heart, diseases of.
FOLLICULITIS. *See* Industrial diseases.
Fomentations, hot, in renal colic, 598
FOREIGN BODIES IN BRONCHI. *See* Bronchi, diseases of.
FORIEGN BODIES IN AIR PASSAGES. *See* Bronchi, diseases of.
FOREIGN BODY IN ESOPHAGUS. *See* Bronchi, diseases of.
Formalin, in endocarditis, 213
Formin, in pyelitis, 586
Fowler position, in pericarditis, 190
Fowler's solution, in Oroya fever, 830
FRAMBESIA TROPICA. *See* Tropical diseases.
Freund's theory and operation, in pulmonary emphysema, 115
Fulguration and radium, in bladder tumor, 650
FUNCTIONAL NEUROSES OF BLADDER. *See* Bladder, diseases, 619

Gallic acid, in hematuria, 504
Galvanocautery, in pericarditis, 194
GANGRENE, JUVENILE. *See* Thromboangiitis obliterans.
GANGRENE OF LUNGS. *See* Lungs, diseases of.
GASOLENE, as industrial poison, 675
Gastric lavage, in snake poisoning, 892
GASTRO-INTESTINAL DISTURBANCES, in renal tuberculosis, 614
Gatch bed, in pyelitis, 582
GEISBÖCK'S DISEASE, 417
Glandular therapy, in osteomalacia, 493
GLASS WORKERS' CATARACT, 718
GLOSSINA PALPALIS, in transmission of trypanosomiasis, 855
Glucose, in bronchopneumonia, 105
GNATHOSTOMA SIAMENSE. *See* Tropical diseases, nematode infection.
Gomenol oil instillation, in tuberculous cystitis, 619
GONORRHEAL CYSTITIS. *See* Bladder, diseases of, cystitis.
GOUT, chronic bronchitis and, 53
Green citrate of iron, in chronic atrophic laryngitis, 25
GUINEA WORM. *See* Tropical diseases, filariasis.

Gymnastics in, movable kidney, 503
— myositis, 484
— postoperative treatment of adhesive pericarditis, 206

Hahn's operation, for movable kidney, 503
Hayem's serum, in Oroya fever, 830
HEADACHE, in chronic nasal sinusitis, 13
HEART, diseases of, 189
— — acute coronary obstruction, 285
— — — references, 292
— — — symptoms, 286
— — — treatment, 287
— — — — drugs, 287
— — — — rest in bed, 287
— — angina pectoris, 272
— — — aortitis, syphilitic, and, 272
— — — atherosclerosis of aorta and coronary arteries and, 272
— — — causation, 272
— — — coronary obstruction and, 272
— — — diagnosis, 274
— — — myocarditis, acute, and, 272
— — — neurotic states and, 272
— — — pericarditis, acute, and, 272
— — — prognosis, 275
— — — prophylaxis, 276
— — — pseudo, 284
— — — — causes, 284
— — — references, 292
— — — symptoms, 274
— — — toxic states and, 272
— — — treatment, 284
— — — — active, 277
— — — — — between attacks, 279
— — — — — — diet, 281
— — — — — — drugs, 281
— — — — — — rest in bed, 282
— — — — — — surgery, 283
— — — — — during attack, 278
— — — — — — drugs, 278
— — — — — — mustard plaster, 279
— — — — individualization, 278
— — cardiac neuroses, 312
— — — irritable heart, 312
— — — — confused with hyperthyrodism, 316
— — — — constitutional type, 313
— — — — diagnosis, 315
— — — — — X-ray, 315
— — — — infectious type, 313
— — — — prognosis, 316
— — — — symptoms, 314
— — — — treatment, 317
— — — — — active, 317
— — — — — baths, 319
— — — — — diet, 319
— — — — — drugs, 319
— — — — — exercise, 318
— — — — — occupation, 319
— — — — — prophylactic, 317
— — — — — psychotherapy, 317
— — — — — sports, 318
— — — — — surgical treatment of foci of infection, 319
— — — references, 320, 321
— — cardiac pain, 288

HEART, diseases of, cardiac pain, diagnoses of conditions causing, 289
——— diseases marked by, 288
——— myocarditis, acute, with, 290
———— diphtheria and, 290
———— rheumatism and, 290
———— scarlatina and, 290
———— treatment, 290
———— typhoid fever and, 290
——— origin, 288
——— pericarditis causing, 289
———— treatment, 290
———— *See also* Pericardium, diseases of.
——— tachycardia, paroxysmal, with, 291
——— valvular heart disease with, 291
———— treatment, 291
—— congenital cardiac disease, 322
——— advance in understanding of, 323
——— clinical classification, 332
——— cyanotic cases, 323, 332
———— clinical classification, 332
———— etiology, 326
———— oxygen unsaturation in, 327
———— pathogenesis, 325
———— tests of cardiac function, 347
———— treatment, 342, 346
————— bloodletting, 342
————— character building, 346
————— diet, 346
————— Dr. Bronson's statement, 346
————— drugs, 342
————— hygiene, 346
————— institutional, 347
————— ligature of patent ductus arteriosus, 358
————— oxygen administration, 343, 358
————— prevention of diseases of childhood, 346
————— preventive, 342, 346
————— symptomatic, 342
————— tonsillectomy, 357
——— non-cyanotic cases, 323, 330, 334
———— cardiac toleration, 356
———— clinical classification, 334
———— diagnosis, 349, 355
————— Krogh spirometer, 355
————— methods, 355
———— pathogenesis, 330
———— treatment, 348
————— avoidance of diseases of childhood, 357
————— preventive, 357
————— radium in tonsillar troubles, 358
————— teeth, removal of curious, 358
——— pathogenesis, 325
———— primary cause, 326
———— secondary causes, 328
——— references, 359-362
——— treatment, 336
———— prenatal care, 336
————— alcohol intoxication, 339
————— congenital syphilis, 339

HEART, diseases of, congenital cardiac disease, treatment, prenatal care, prenatal infections and intoxications, 338
————— rheumatic fever, 338
————— small-pox, 339
————— tuberculosis, 338
———— preventive, 339
————— antiluetic, 341
———— surgical, 336, 357
—— disorders of heart beat, 293
——— auricular fibrillation, 301
———— treatment, 302
————— drugs, 302
————— methods, 302
——— auricular flutter, 299
———— treatment, 300
————— digitalization, 300
————— quinidin sulphate, 300
——— extrasystoles, 295
——— heart-block, 308
———— auriculoventricular, 308
————— arteriosclerosis of coronaries and, 308
————— causes, 308
————— digitalis and, 308
————— Stokes-Adams syndrome in, 309
———— intraventricular, 308
————— arteriosclerosis and, 308
————— causes, 308
————— digitalis, 309
———— treatment, 309
————— drugs, 309
————— etiological, 309
————— symptomatic, 309
————— thyroid extract, 309
——— paroxysmal tachycardia, 297
———— atrioventricular, 299
———— causes, 297
———— nodal, 299
———— treatment, 297
————— drugs, 297
————— symptomatic, 297
——— premature contractions, 295
———— causes, 295
———— treatment, 296
————— digitalis, 296
————— rest, 296
——— pulsus alternans, 310
———— treatment, 311
————— digitalis, 311
————— rest, 311
——— sinus arhythmia, 293
——— sinus bradycardia, 294
———— treatment, 294
——— sinus tachycardia, 294
———— treatment, 295
————— rest in bed, 295
—— endocarditis, 207
——— acute bacterial, 219
———— course, 221
———— etiology, 219
———— pathology, 219
———— symptoms, 220
————— Janeway lesion, 220
————— murmurs, 221
————— petechial, 220

Heart, diseases of, endocarditis, acute bacterial, symptoms, preventive, 221
— — — — — serums, 221
— — — — — sodium cacodylate, 221
— — — — — transfusions, 221
— — — — — vaccines, 221
— — — sub-acute bacterial, 207
— — — — diagnosed as malaria, 209
— — — — diagnosis, 210
— — — — — bacteria-free stage, 210
— — — — — bacterial stage, 210
— — — — mistaken for surgical condition, 217
— — — — pathology, 207
— — — — prophylaxis, 218
— — — — — vaccination, 219
— — — — symptoms, 207
— — — — — anemia, 209
— — — — — blood cultures, 210
— — — — — due to infection, 208
— — — — — emboli, 209
— — — — — fever, 208
— — — — — kidney affections, 210
— — — — — myocardial, 208
— — — — — pain and tenderness, 209
— — — — — petechiæ, 209
— — — — — renal phenomena, 212
— — — — — spleen, enlargement of, 210
— — — — treatment, 212
— — — — — chemical antidotes, 213
— — — — — drugs, 213
— — — — — serums, 212
— — — — — transfusions, 214
— — — — — vaccines, 212
— — myocarditis, 222
— — — acute, 222
— — — — arhythmas developing suddenly in, 224
— — — — diphtheria complicated by, 223
— — — — pneumonia and, 223
— — — — primary acute inflammation of heart muscle and, 222
— — — — references, 225, 226
— — — — rheumatism and, 223
— — — — symptomatology, 223
— — — — treatment, 224
— — — — — diet, 224
— — — — — drugs, 224
— — — — — ice-bag, 224
— — — — — rest, 224
— — — — typhoid fever and, 223
— — — chronic, 227
— — — — etiology, 232
— — — — — arteriosclerosis, 232
— — — — — chorea, 232, 261
— — — — — hypertension, 232, 262
— — — — — hyperthyroidism, 232, 262
— — — — — syphilis, 232, 261
— — — — — tonsillitis, 232, 261
— — — — pathological physiology, 227
— — — — — arhythmias, 230
— — — — — pulsus alternans, 231
— — — — — x-ray, 229
— — — — prevention, 261
— — — — references, 266-271
— — — — special conditions, 262
— — — — — angina pectoris, 266
Heart, diseases of, myocarditis, chronic, special conditions, aortic insufficiency, 266
— — — — — auricular fibrillation, 262
— — — — — auricular flutter, 264
— — — — — extrasystoles, 264
— — — — — heart-block, 265
— — — — — high blood-pressure, 265
— — — — — paroxysmal tachycardia, 265
— — — — — pulsus alternans, 265
— — — — symptomatology, 233
— — — — treatment, 233
— — — — — activity and exercise, 259
— — — — — bleeding, 256
— — — — — clothing, 235
— — — — — continuation, 257
— — — — — diet, 256
— — — — — drugs, 236, 251, 258
— — — — — mechanical removal of fluid, 255
— — — — — rest, 234
— — — — — stage of heart failure, 233
— — pericarditis, 188
— — — acute, 191
— — — — diet, 191
— — — acute rheumatic fever and, 188
— — — adhesive, 204
— — — — polyserositis in, 204
— — — — treatment, 204
— — — — — operative, 204
— — — — — postoperative, 206
— — — — — prophylactic, 204
— — — dysphagia and, 189
— — — glands of the neck and, 189
— — — hemopericardium, 203
— — — — treatment, 203
— — — hydropericardium, 201
— — — — cachetic conditions and, 201
— — — — chronic nephritis and, 201
— — — — scarlet fever and, 201
— — — — subacute inflammation of endothelial cells and, 201
— — — — treatment, 202
— — — — — adrenalin, after paracentesis, 202
— — — — — diuretics in, 202
— — — — — paracentesis in, 202
— — — — — sterilized air after removal of pericardial effusion, 202
— — — — tuberculosis and, 201
— — — — tumor of the pericardium and, 201
— — — pneumopericardium, 204
— — — — treatment, 204
— — — prevention of vomiting in, 195
— — — prophylaxis, 188
— — — pyopericardium, 203
— — — — purulent pleurisy and, 203
— — — — treatment, 203
— — — tonsils and, 189
— — — treatment, 191
— — — — after, 197
— — — — — carbonic acid baths, 197
— — — — — gymnastics, 197
— — — — cough, 191
— — — — — diuretics, 198
— — — — drugs, 191, 194
— — — — — digitalis, 196

HEART, diseases of, pericarditis, treatment, drugs, morphin, 196
— — — — — salicylates, 195
— — — — — sodium cacodylate, 195
— — — — enema, 191
— — — — Fowler position, 190
— — — — local, 193
— — — — — blisters, 194
— — — — — bloodletting, 194
— — — — — cold applications, 193, 196
— — — — — galvanocautery, 194
— — — — — Paquelin cautery, 194
— — — — myocarditis, 191
— — — — rest in bed, 190
— — — — stimulants, 196
— — — — — strophanthin, 197
— — — with effusion, 197
— — — — treatment, 197
— — — — — paracentesis, 198
— — *See also* Blood vessels, diseases of.
HEART, in acute infectious diseases, 222
— *See also* Heart, diseases of, myocarditis, acute.
HEART, IRRITABLE, 312.
— — *See also* Heart, diseases of.
HEART BEAT, 293.
— *See also* Heart, diseases of.
HEART-BLOCK, 308
— *See also* Heart, diseases of.
HEAT CRAMPS, 709
HEAT EXHAUSTION, 707-709
HEAT, in occupational diseases. *See* Industrial diseases.
HEAT STROKE, 707
Heliotherapy in, paronychia, 794
— ulcer, 796
Helmitol, in pyelitis, 586
HELODERMA. *See* Vertebrates, poisonous.
HEMATURIA in, renal tuberculosis, 609, 613
— tuberculous kidney, 600 613
HEMATURIA. *See also* Hemorrhages in essential hypertension.
HEMATOCHYLURIA, treatment of, 881
HEMOPERICARDIUM, 201
HEMOPTYSIS, 417
HEMORRHAGES, in essential hypertension, 412, 416
HEMORRHAGIC PLEURISY, 163
HEMOTHORAX, 163
Heroin hydrochlorid in bronchitis, 43
Hexamethylenetetramin, in pyelitis, 586
HIGH BLOOD-PRESSURE STASIS. *See* Essential hypertension.
High frequency in bladder tumor, 636
HILL DIARRHEA. *See* Tropical diseases.
Hofbauer's expirator, in pulmonary emphysema, 118
HUMIDITY, in occupational diseases. *See* Industrial diseases.
HUMIDITY AND HEAT, in occupational diseases, 710-714
HUNNER ULCER. *See* Bladder, diseases of.
Hychlorite, in industrial infections, 659
HYDRASTIN, effect on pulmonary pressure of, 90
Hydrastinin hydrochlorid, in epistaxis, 18
HYDROCHLORIC ACID, as industrial poison, 287
HYDROFLUORIC ACID, in industrial poison, 687
Hydrogen peroxid, in industrial infections, 659
Hydromineral treatment of phlebitis, 467
HYDRONEPHROSIS. *See* Kidney, diseases of.
HYDROPERICARDIUM in, cardiac failure with edema, 409
— pericarditis, 201
Hydrotherapy in, arteriosclerosis, 370, 375
— insulation, 810
— occupational diseases, 708
— pleurisy, 147, 162
— renal colic, 598, 599
— renal tuberculosis, 612, 613
Hygiene, in bronchopneumonia, 105
— pulmonary emphysema, 116
Hyoscyamus, in endemic hematuria, 879
— enuresis, 626
HYPEREMIA. *See* Congestion of lungs, active.
HYPERESTHESIA, in occupational neuroses, 779
HYPERPIESIA, ESSENTIAL, 378
— — *See also* Blood vessels, diseases of.
HYPERTENSION, ESSENTIAL, 378
— — in etiology of chronic myocarditis, 232
— — *See also* Blood vessels, diseases of.
HYPERTENSIVE type of arteriosclerosis, 377
HYPERTHYROIDISM, in chronic myocarditis, 232
Hypcdermocylsis, in suppression of urine, 603
HYPOSTATIC CONGESTION. *See* Lungs, diseases of.

Ichthyol, in ozena, 19
Ichthyol ointment, in paronychia, 794
ILLUMINATION, ARTIFICIAL, 730-732
Immobilization, in phlebitis, 463
IMMUNITY, in yellow fever, 847
Imperial drink, in suppression of urine, 603
Implantation of radium seeds, in bladder tumors, 648
Incandescent light baths, in chronic bronchitis, 60
Incision and drainage, in pyelitis, 588
INDURATIVE MEDIASTINOPERICARDITIS. *See* Mediastinopericarditis, indurative.
INDUSTRIAL diseases, 653
— caisson-disease, 690
— — aural disturbances and, 693
— — caissons, 690, 693
— — — air-pressure in, 691
— — cause of, 690
— — — air-pressure, 697, 698
— — — — degrees, 697, 698
— — — duration of working shifts, 697, 698
— — — predisposing factors, 696, 697
— — — theories, 691, 692

INDUSTRIAL diseases, caisson-disease, complications of, 696
—— decompression in, 701
——— oxygen and, 703
——— methods, 701, 703
——— rate of, 702, 703
—— diving and, 691
—— history of, 690
—— immunity, 701
—— individual susceptibility, 700
——— age, 700
——— obesity, 700
—— mortality, 694
—— occurrence, 693, 694
—— pathology, 696
—— references, 767
—— symptoms, 694
——— collapse, 695
——— dyspnea, 695, 696
——— nervous system disorders, 695
——— onset, 693
——— pain, 694, 695, 705
——— vertigo, 695
—— treatment, 704
——— medical, 705
———— symptomatic, 706
——— recompression, 704
———— degree of air pressure, 706
———— results, 705, 706
—— ventilation, 699, 700
— cold and, 714
—— effects of, 714, 715
—— moisture and, 714, 715
—— prophylaxis, 715
—— references, 768
—— treatment, 715
— electricity and, 741
—— burns, 742
——— treatment, 743
—— references, 770
—— shock, 743
——— after-effects, 746
——— death and, 743
——— degrees, 743
——— effects, 743, 744
——— lightning, 747
———— effects, 747
——— prophylaxis, 747
——— treatment, 744-746
—— types of currents, 741
— fatigue and, 748
—— definition, 748
—— industrial, 755
——— apparatus and, 766
——— humidity and, 764
——— lighting and, 764
——— night work and, 764
——— occupational neuroses, 766
——— temperature and, 764, 765
——— tests, 752
———— Martin's spring balance, 752
———— Ryan reaction, 752
———— Schneider, 752
————— technic, 752, 753, 754
——— treatment, 767
——— ventilation and, 764
——— workers' output, 755-761

INDUSTRIAL diseases, fatigue and, industrial, workers' output, hours of work and, 761, 762
———— rest periods and, 763
—— physiology of, 748-752
—— references, 770, 771
— heat and, 706
—— effects of, 706, 709, 710
—— heat cramps, 709
——— onset, 709
——— symptoms, 709
——— treatment, 709
—— heat exhaustion, 707, 708
——— symptoms, 708
——— treatment, 708
——— *See also* Insolation.
—— heat stroke, 707
——— prognosis, 708
——— treatment, 709
——— *See also* Insolation.
—— humidity and, 710
——— effects, 710-714
——— references, 768
—— prophylaxis, 710
—— references, 768
—— *See also* Insolation.
— infections, 653
—— extrahazardous industries and, 654
—— neglect of slight injuries and, 654
—— occupations and, 654
—— prevention of, 656
——— abrasions or wounds covered with grease or dirt, 659
———— treatment, 659
——— antiseptics, 658
———— alcohol, 659
———— aristol, 659
———— bichlorid of mercury solution, 659
———— boric acid, 659
———— cresol, 659
———— dichloramin-T, 658, 659
———— hychlorite, 659
———— hydrogen peroxid, 659
———— iodin, Churchill's tincture of, 659
————— tincture of, 659, 660
———— iodoform, 659
———— picric acid, 659
———— zinc oxide, 659
——— dressings, 659
———— alcohol and glycerine, 659
———— balsam of Peru with castor oil, 659
———— iodoform, 659
——— first-aid kits, 657
———— Factory Department of British Home Office instructions, 657, 660
———— iodin, 657, 660
———— printed directions, 657
———— sterilized dressings, 657, 660
——— splinters of wood or metal, 658
——— scabs, removal of, 659
— intoxications, 661
—— bacterial, 662
——— individual susceptibility, 662
———— age, 662
———— alcohol, 662

Industrial diseases, intoxications, bacterial, individual susceptibility, poor nutrition, 662
— — — — sex, 662
— — diagnosis of, 662
— — dust, 661
— — entry and exit of, 661
— — fumes, 661
— — liquid, 661
— — metals and their compounds, 663
— — — antimony, 663
— — — — pathology, 663
— — — — prevention, 664
— — — — symptoms, 663
— — — — treatment, 664
— — — arsenic, 664
— — — — acute, 664
— — — — — symptoms, 664
— — — — — treatment, 664
— — — — chronic, 665
— — — — — symptoms, 665
— — — — — treatment, 665
— — — — pathology, 664
— — — — prevention, 665
— — — — symptoms, 666
— — — — treatment, 664
— — — — — drinking water, 665
— — — — — milk diet, 665
— — — — — morphin, 665
— — — — — oxygen inhalation, 665
— — — brass—zinc—etc., 665
— — — — acute, 666
— — — — — symptoms, 666
— — — — brass founder's ague, 665
— — — — — diagnosis, 666
— — — — — malaria, resemblance to, 666
— — — — — prevention, 667
— — — — — treatment, 667
— — — — chronic, 667
— — — — — arteriosclerosis and, 667
— — — — — bronchitis and, 667
— — — — — cause, 667
— — — — — emphysema and, 667
— — — — — fibrosis of lungs and, 667
— — — — — prevention, 667
— — — — — symptoms, 667
— — — — — treatment, 667
— — — — — tuberculosis of lungs and, 667
— — — lead, 667
— — — — arteriosclerosis and, 668
— — — — ceramic industry workers and, 668
— — — — diagnosis, 671
— — — — encephalopathy and, 668, 669
— — — — gout and, 668
— — — — interstitial nephritis and, 668
— — — — lesions of brain and meninges and, 668
— — — — menorrhagia and, 669
— — — — neuritis and, 668
— — — — painters and, 668
— — — — pathology, 668
— — — — — age, 668
— — — — — alcohol, 668
— — — — — sex, 668
— — — — prevention of, 670
— — — — smelters and, 668

Industrial diseases, intoxications, metals and their compounds, lead, symptoms, 668
— — — — — anemia, 669
— — — — — arteries thickened, 670
— — — — — atrophy and fibrillary tremors, 669
— — — — — blindness, 669
— — — — — blood-pressure, high, 670
— — — — — cerebral, 669
— — — — — convulsions, 669
— — — — — heart enlarged, 670
— — — — — lead line, Burtonian, 670
— — — — — leukocytosis, 669
— — — — — menorrhagia, 669
— — — — — nephritis, 670
— — — — — parasthesia, 669
— — — — — weakness or paralysis of muscles, 669
— — — — — wrist-drop, 670
— — — — treatment, 671
— — — — — anemia, 671
— — — — — colic, 671
— — — — — paralysis, 671
— — — — white lead makers and, 668
— — — manganese, 672
— — — — pathology, 672
— — — — prevention, 672
— — — — symptoms, 672
— — — — treatment, 672
— — — — — diet, 672
— — — — — hydrotherapy, 672
— — — — — laxatives, 672
— — — — — massage, 672
— — — — — rest, 672
— — — — — sweating, 672
— — — mercury, 672
— — — — pathology of, 673
— — — — prevention, 674
— — — — symptoms, 673
— — — — — esethism, 673
— — — — — stomatitis, 673
— — — — — tremor, 673
— — — — treatment, 674
— — miscellaneous, 687
— — — acetaldehyd, 687, 688
— — — acridin, 687, 688
— — — acrolein, 687, 688
— — — ammonia, 687, 688
— — — bromin, 687, 688
— — — cadmium, 689
— — — chlorin, 687, 688
— — — chromium compounds, 687
— — — copper, 689
— — — cyanids, 687
— — — cyanogen, 687
— — — diethylsulphate, 687, 688
— — — dimethylsulphate, 687
— — — fluoric acid, 687
— — — hydrochloric acid, 687, 688
— — — hydrofluoric acid, 687, 688
— — — iodin, 688
— — — methyl bromid, 688
— — — methyl sulphate, 688
— — — nicotin, 688
— — — nitric acid, 687, 688
— — — phosgine, 687

INDUSTRIAL diseases, intoxications, miscellaneous, prussic acid, 687
——— sulphur dioxid, 687
——— sulphuric acid, 687, 688
——— tellurium, 689
—— non-metals and their compounds, 674
——— amino compounds of benzol and its homologues. *See* Anilin.
——— anilin, 674, 688
———— diagnosis, 675
———— mistaken for valvular heart disease, 675
———— pathology, 674
———— prevention, 675
————— alcohol prohibited, 675
————— hygiene, 675
————— medical examinations, 675
————— protection, 675
———— symptoms, 674, 688
———— treatment, 675
————— artificial respiration, 675
————— blood transfusion, 675
————— heart stimulation, hypodermic, 675
————— laxatives, 675
————— oxygen inhalations, 675
————— venesection, 675
——— benzene — benzin — naphtha — gasoline, 675, 688
———— pathology, 676
———— prevention, 676
———— rest, 677
———— symptoms, 676, 688
———— treatment, 677
————— artificial respiration, 677
————— blood transfusion, 677
————— fresh air, 677
————— oxygen inhalations, 677
——— carbon bisulphid, 677, 688
———— acute, 677
————— pathology, 677
————— symptoms, 677
———— chronic, 677
————— symptoms, 677, 688
———— prevention, 678
———— treatment, 678
——— carbon monoxid, 679, 688
———— acute, 679
———— chronic, 679
———— pathology, 679
———— prevention, 680
———— treatment, 680
————— artificial respiration by Shaeffer method, 680
————— food, 680
————— fresh air, 680
————— rest, 680
————— warmth, 680
——— carbon tetrachlorin, 680, 688
———— prevention, 680
———— symptoms, 680, 688
———— treatment of, 680
————— artificial respiration, 680
——— methyl alcohol, 681, 688
———— chronic, 681
———— pathology, 681
———— prevention, 681

INDUSTRIAL diseases, intoxications, non-metals and their compounds, methyl alcohol, symptoms, 681, 688
———— treatment of, 681
————— artificial respiration, 681
————— bleeding, 681
————— blood transfusion, 681
——— nitrobenzol, 681, 688
———— pathology, 681
———— prevention, 682
———— symptoms, 681, 688
———— treatment, 682
————— artificial respiration, 682
————— heart stimulation, 682
————— oxygen inhalation, 682
——— nitroglycerin, 682, 688
———— pathology, 682
———— prevention, 683
———— symptoms, 682, 688
———— treatment, 683
——— phosphorus, 683, 688
———— pathology, 683
———— prevention, 683
———— symptoms, 683, 688
———— treatment, 683
————— artificial respiration, 683
——— sulphurated hydrogen, 683, 688
———— pathology, 683
———— prevention, 684
———— symptoms, 683, 688
———— treatment, 684
————— artificial respiration, 684
————— blood transfusion, 684
——— tetrachlorethane or tetralin, 684, 688
———— pathology, 684
———— prevention, 685
———— symptoms, 684, 688
——— trinitrotoluene, 685, 688
———— pathology, 685
———— prevention, 686
———— symptoms, 685, 688
———— treatment, 686
——— turpentine, 686
———— pathology, 686
———— prevention, 687
———— symptoms, 687
———— treatment, 687
————— nephritis, 687
—— pathology of, 661
—— prevention of, 662
—— references, 689
—— symptoms of, 661
—— table of industrial poisons and their effects, 688
—— treatment of, 663
— light and, 716
—— analyses of, 717
—— eclipse blindness, 725
——— cause, 725
——— prophylaxis, 725
—— effects of, 718
—— electric ophthalmia, 720
——— cause, 721
——— incidence, 721
——— symptoms, 721
——— treatment, 721

INDUSTRIAL diseases, light and, eyestrain, 729
———artificial lighting and, 730
————types, 731
———causes, 730
———incidence, 729
———prophylaxis, 732
——glass workers' cataract, 718
———cause, 718
———incidence, 718
———prophylaxis, 719
———treatment, 719, 720
——miners' nystagmus, 727
———cause, 727, 728
———complications, 729
————psychic, 729
———incidence, 727
———symptoms, 727
———treatment, 729
——ocular fatigue, 725
———cause, 726
———prophylaxis, 726
———symptoms, 726
——references, 768, 769
——sources of, 717
——ultraviolet rays, 721
———eyes and, 721
————effect, 721, 723
————treatment, 724, 725
———metabolism and, 724
———protection against, 724
————effect, 721, 723
———sunlight and, 721
————intensity, 721
—————variation, 722
——waves, 716
—occupational dermatoses, 782
——acne, 784
———etiology, 784
———prophylaxis, 784
———treatment, 785
——actinomycosis, 785
———etiology, 785
———prophylaxis, 785
———treatment, 785
——anthrax, 786
———etiology, 786
———pathology, 786
———treatment, 786
————serum, 786
——burns, 786
———etiological factors, 786
————incidence, 787
———prophylaxis, 787
———treatment, 787
——callositas, 787
———etiological factor, 787
———prophylaxis, 787
———treatment, 787
——cutaneous tuberculosis, 795
———etiology, 795
———prophylaxis, 795
———treatment, 796
——dermatitis, 788
———acute, 788
————etiological factors, 788
————prophylaxis, 788
INDUSTRIAL diseases, occupational dermatoses, dermatitis, acute, treatment, 788, 789
——dermatitis factitia, 793
———treatment, 793
——eczema, 789
———etiological factors, 790
———prophylaxis, 790
———treatment, 790, 791
————medical, 790
———treatment, 791
————x-ray, 791
——epithelioma, 791
———etiological factors, 791
———prophylaxis, 791
———treatment, 791
————surgical, 791
————x-ray, 791
——etiology of, 782
——folliculitis, 791
———etiological factors, 791
———prophylaxis, 791
———treatment, 792
————black wash, 792
————x-ray, 792
——keloids, 792
———etiological factors, 792
———prophylaxis, 792
———treatment, 792
————radium, 792
————x-ray, 792
——paronychia, 793
———etiological factors, 793
———prophylaxis, 793
———treatment, 793
————heliotherapy, 794
————ichthyol, 794
————x-ray, 794
——prophylaxis, 783, 784
——pus infections, 794
———etiological factors, 794
———prophylaxis, 794
———treatment, 794
————medical, 794
————surgical, 794
——susceptibility, 782
——tinea, 795
———fungus infection, 795
———prophylaxis, 795
———treatment, 795
——treatment of, 784
——ulcer, 796
———etiological factors, 796
———prophylaxis, 796
———treatment, 796
————heliotherapy, 796
————medical, 796
—occupational neuroses, 777
——causes of, 776, 777
——diagnosis of, 779
———differential, 779
————functional neurosis, 779
————hysteria, 779
——incidence, 777
———sex, 777
——prognosis, 780
——symptoms, 777-780

INDUSTRIAL diseases, occupational neuroses, synonyms, 776
——treatment of, 780, 781
—pneumoconiosis, 772
——anthracosis, 772
——diagnosis, 773
———differential, 773
————tuberculosis, 773
———x-ray, 773
——dust in, 772, 773
———action of, 773
——etiology of, 772
——incidence, 772
——onset, 773
——prognosis of, 773
——prophylaxis, 774, 775
——references, 775
——siderosis, 772
——silicosis, 772, 773
——stages of, 774
——symptomatology, 773
———dyspnea, 773
——treatment of, 774
——tuberculous infection and, 773, 774
—radiation and, 732
——radium, 732
———action, 735
———dermatitis and, 737, 738
————prophylaxis, 740
———dosage, 736
———emanations, 737
———ions, 736
———sensitivity, 736, 737
———sterility and, 737
———types of rays, 735
————alpha, 735
————beta, 735
————gamma, 735
——references, 769
——x-rays, 732
———action, 735
———dermatitis and, 738, 739
————prophylaxis, 739, 740
————treatment, 740
———discovery, 732
———effects, 732
———production, 733
————Coolidge tube, 734
————Crookes' tube, 734
———short waves, 734, 735
———sterility and, 737
———voltage, 734
INDUSTRIAL INFECTIONS. *See* Industrial diseases.
INDUSTRIAL INTOXICATIONS. *See* Industrial diseases.
INDUSTRIAL POISONS. *See* Industrial diseases.
INFECTIONS, INDUSTRIAL. *See* Industrial diseases.
INFECTIOUS TYPE OF ARTERIOSCLEROSIS, 347
INFLUENZA, producing hyperplastic ethmoiditis, 16
INFLUENZA PNEUMONIA, EPIDEMIC, and pulmonary emphysema, 113
Injections, intrapleural, in pleurisy, 160
—intratracheal, in bronchiectasis, 66
Injections, intravenous in thrombo-angiitis obliterans, 477
—subcutaneous in thrombo-angiitis obliterans, 477
INSECTS, POISONOUS. *See* Poisons—reptiles—fish—arthropods and plants, affections due to.
INSOLATION, 804
—apyrexial, 811
——treatment, 811
—body heat loss, 806
——table by radiation and conduction, 806
—causes of, 805
——radiation, 807
———experiments showing, 807
——tropical climates, 806
——violet and ultraviolet rays, 805
——water loss, 808
—classification of, 805
—prophylaxis of, 808
—references, 811
—treatment of, 809
——drugs, 810
——hydrotherapy, 810
——ice-cold packs or baths, 810
——saline solutions, infusion of, 810
——stimulants, 810
INSOMNIA, in chronic bronchitis, 55
Institutional treatment, in congenital heart disease, 347
INTERMITTENT CLAUDICATION, in arteriosclerosis, 376
—*See also* Thrombo-angiitis obliterans.
INTESTINAL FLUKES, 875, 876
INTESTINAL PARASITES, 871, 872, 879
Intubation, in membranous laryngitis, 25
Iodid of iron, in chronic bronchitis, 53
Iodid of potassium in, angina pectoris, 282
—plastic bronchitis, 60
Iodids in, arteriosclerosis, 372
—bronchopneumonia, 104
—embolism, 457
—phlebitis, 467
—pleurisy, 148
—syphilitic pleurisy, 143
—thrombo-angiitis obliterans, 475
—tuberculosis of mediastinal lymph nodes, 179
IODIN, as industrial poison, 688
Iodin in, atrophic rhinitis, 8
—ozena, 19
—syphilis of the upper respiratory tract, 30
—tinea, 795
Iodipin in chronic bronchitis, 54
Iodoform, in industrial infections, 659
Iodoform in oil, instillations of, in tuberculous cystitis, 619
Ionatherapy. *See* Ionization.
Ionization, in dermal leishmaniasis, 866
—potassium iodid, in thrombosis, 448
Iontophoresis. *See* Ionization.
Ipecac, in spasmodic laryngitis, 23
Ipecacuanha, in plastic bronchitis, 61
IRIDOCYCLITIS, in trypanosomiasis, 849

Iron in, arteriosclerosis, 373
—lead poisoning, 671
—nephritis, 523
—Oroya fever, 830
—phlebitis, 465
Irrigation of bowel, in pyelitis, 584, 585
IRRITABLE HEART, 312
—*See also* Heart, diseases of.
IRRITATIVE POISONOUS PLANTS. *See* Poisons—reptiles—fish—arthropods and plants, affections due to.

Jackson tubes, in syphilis of the upper respiratory tract, 31
Janet syringe, in bladder tumors, 535
JANEWAY LESION, in diagnosis of acute bacterial endocarditis, 220
JIGGER, POISONING by, 900
JUVENILE GANGRENE. *See* Thrombo-angiitis obliterans.

KALA AZAR. *See* Leishmaniasis, visceral.
Karell diet, before transfusion for endocarditis, 215
Karell diet in, accidents to cerebral vessels, 413
—chronic myocarditis, 256
KATAYAMA disease. *See* Tropical diseases, schistosomiasis.
KELOIDS. *See* Industrial diseases.
KIDNEY, abscess of. See Kidney, diseases of.
—diseases of, 508
——amyloid disease of kidney, 576
———causes, 576
————chronic suppurative processes, 576
————syphilis, 576
————tuberculosis with secondary pyogenic infection, 576
———parenchymatous nephritis and, 576
———pregnancy, kidney of, 577
————treatment, 577
—————cathartics, saline, 578
—————cesarean section, 578
—————diet, 577
—————drinking water, 577
—————drugs, 577
—————elimination increased, 577
—————oxygen inhalation, 578
—————protein food reduced, 577
—————sweating, 577
—————uterus emptied, 578
—————venesection, 577
———references, 578
———treatment, 576
————neurasthenic symptoms, 577
—————psychotherapy, 577
————prophylactic, 576
—————antisyphilitic, 576
—————drainage of pus collections, 576
—————diet, 576
—————rest, 577
————*See also* Toxemias of pregnancy, III, p. 3
KIDNEY, diseases of, anuresis, 503
———causes, 503
———definition, 503
———types, 503
————cardiac decompensation causing, 504
—————treatment, 504
————hysterical, 504
—————treatment, 504
————infectious processes causing, 504
—————treatment, 504
————nephritis causing, 503
—————treatment, 503
————obstructive, 503
—————treatment, 503
————reflex, 50
—————treatment, 504
————toxemia causing, 504
——hematuria, 504
———causes, 504
————neoplasm in genito-urinary tract, 504
————hemostatics, 504
———treatment, 504
————diet, 504
————nephrotomy, 504
——hemoglobinuria, 504
———paroxysmal, 506
————causes of, 506
—————exposure to low temperature, 506
—————syphilis, 506
————malaria and, 507
—————treatment, 507
————prevention, 506
————treatment, 507
—————antisyphilitic, 507
—————Wassermann test and, 507
———toxic, 504
————causes, 505
—————coal-tar drugs, 505
—————hydrogen arsenic inhalation, 505
—————hydrochloric acid intoxication, 505
—————potassium chlorate intoxication, 505
—————soap bark ingestion, 505
—————solanin ingestion, 505
—————sulphuric acid intoxication, 505
—————uncooked mushrooms ingestion, 505
————clinical picture, 505
————hematoporphyrinuria, 504
—————treatment, 506
————hemolysis in, 505
————malaria and, 505
————methemoglobinuria, 504
—————treatment, 506
————purpura and, 505
————scarlatina and, 505
————scurvy and, 505
————syphilis and, 505
————treatment, 506
————typhoid fever and, 505

Kidney, diseases of, hemoglobinuria, toxic, typhus and, 505
———— yellow fever and, 505
—— hydronephrosis, 592
——— acquired, 594
———— diagnosis, 594
————— cystoscopy, 594
————— x-ray, 594
——— causes, 592
——— congenital, 593
———— bilateral, 593
———— unilateral, 593
————— treatment, 593
——— diagnosis, 593
——— forms, 592
———— acquired, 592
———— congenital, 592
——— intermittent, 593
———— symptoms, 593
———— tumor, 593
——— lesions in, 592
——— references, 595
——— results of, 592
——— treatment, 593
———— kidney function and, 594, 595
———— surgical, 594, 595
————— contra-indications, 594
————— indications, 594, 595
——— types of, 593
———— intermittent, 593
———— permanent, 593
—— uremia in, 595
—— lithiasis, 604
——— alcohol, 605
———— prohibition, 605
——— alkalis, 606
——— alkaline waters, 605, 606
——— calculus, 604
———— oxalates, 604
———— phosphates, 604
———— uric acid, 604
——— diet in, 604, 605
——— exercise in, 604
——— metabolism, 604
——— oliguresis, 605
—— movable kidney, 501
——— abnormal, 501
———— causes of, 501
————— dislocation, 502
———— hysterical cases, 502
———— neurasthenic cases, 502
———— treatment, 502
————— bandaging, 502
————— corset, supporting, 502
————— diet, 502
————— electricity, 503
————— gymnastics, 503
————— massage, abdominal, 503
————— rest, 502
——— normal, 501
—— nephritis, 508, 522
——— acute, 551
———— prophylaxis, 552
————— alcoholic drinks, 552
————— cold, 552
————— diet, 552
————— infections, 552

Kidney, diseases of, nephritis, acute, prophylaxis, irritants, kidney, 552
———— treatment, 553
————— active, 553
————— alkalis, 554
————— anemia, 558
————— anuresis, 557
————— backache, 558
————— baths, cold, avoided, 553
————— catharsis, 553
————— decapsulation, 556
————— diet, 554
————— diuretics, 553
————— dyspnea, 558
————— edema, 557
————— edema of glottis, 558
————— fluid intake, 556
————— gastric disturbances, 558
————— headache, 558
————— hemorrhagic nephritis, 559
————— itching, 558
————— rest, 553
————— sodium chlorid, 556
————— special, 551
————— sweating, 556
————— uremia, 557
——— chronic interstitial, 523, 561
———— etiology, 562
———— treatment, 562
————— catharsis, 565
————— cerebral hemorrhage, 565
————— diet, 562
————— digitalis, 565
————— dyspnea, 564
————— elimination, increased, 563
————— headache, 565
————— heart, 523
————— hypertension, 563
————— morphin, 565
————— salt restriction, 562
————— sweating, 563, 565
————— vasodilators, 563
————— venesection, 564, 565
——— chronic parenchymatous, 523, 559
———— bowel and skin elimination, 560
———— diet, 560
———— diuretics, 560
———— etiological factors, 559
——— etiology, 523, 552
———— alcohol, 524, 552
———— chemical poisons in, 523
———— cold, 523, 525, 552
———— drugs, 524, 552
———— infections, 523, 552
———— occupations, 523
———— pregnancy, 523
———— syphilis, 524, 552
——— hypertension, 540
———— treatment, 540
————— reduction by increased elimination, 541
————— vasodilators, 542
————— venesection, 542
——— mercurial, 566
———— treatment, 567

KIDNEY, diseases of, nephritis, mercurial, treatment, irrigations, rectal, 567
—————— stomach, 567
————— whites of eggs, 567
——— prophylaxis, 523, 552
——— references, 567-575
——— treatment, 522
———— alcohol, 539
———— bathing, 528
———— bowel regulation, 523
———— catharsis, 529
———— climate, 523, 526
———— clothing, 523
———— diet, 522, 536, 552
————— coffee, 539
————— daily food requirement, 537
————— milk, 538
————— protein, 536, 538
————— tea, 539
———— diuretics, 544
———— fluids, restriction of, 530
———— general principles, 522
———— iron, 523
———— massage, 528
———— rest, 527, 552
———— salt, amount in average portion of foods, 533
———— skin, care of, 528, 552
———— sodium chlorid, content of foods, 533
————— restriction of, 531
———— sweating, 533
————— methods, 535
————— shock, danger of, 534
———— symptomatic, 522
———— tobacco, 539
——— uremia, 547
———— treatment, 547
————— diet, 547
————— diuretics, 549
————— drainage by puncture of legs, 549
————— drugs, 549
————— elimination through bowel and skin, 547
————— lavage of stomach, 549
————— venesection, 548
————— vomiting, 549
——— uremic dyspnea and pulmonary edema, 550
———— treatment, 550
————— alkalis, 550
————— drugs, 550
————— sweating, 551
————— vasodilators, 550
————— venesection, 550
—— nephrolithiasis, 596
——— infection in, 603
———— results, 603
———— symptoms of, 597
—— pyelitis, 579
——— complications, 589
———— cardiac hypertrophy and hypertension, 589
————— treatment, 589
———— cystitis, 589

KIDNEY, diseases of, pyelitis, complications cystitis, treatment, 589
———— gastro-intestinal symptoms, 589
————— treatment, 590
———— peritonitis, 589
———— retention of urine, 589
————— treatment, 589
———— tuberculosis of kidney, 589
——— defloration, 580
——— diagnosis, 581
———— tests, 581, 583, 588
———— x-ray examination, 581
——— etiology, 579
———— bladder spasm, 580
———— colon inflammation, 580
———— constipation, 580
———— cystitis, 580
———— growth in pelvis or ureter, 580
———— hydronephrosis, 580
———— inflammation or blockage, 580
———— local lesions, 580
———— new growths, 580
———— organisms responsible for, 579
———— prostatic enlargement, 580
———— renal calculus, 580
———— routes of invasion, 579
———— strictures of urethra, 580
———— tuberculous kidney, 580
——— prophylaxis, 590
——— pyelonephritis, 580
———— calculus and, 580
———— cystitis and, 580
———— diagnosis, 581
———— grip and, 580
———— pneumonia and, 580
———— prostatitis and, 580
———— renal calculus, 589
———— symptoms, 580
———— typhoid and, 580
——— references, 591
——— symptoms, 581
——— treatment, 581
———— alkaline salts, 585
———— antiseptics, urinary, 584
———— bathing, 582
———— benzoic acid, 585
———— bowel regulation, 584
————— cathartics, 584
————— irrigation, 584, 585
———— diet, 582
————— caloric requirement, 582
————— fats and carbohydrates, 583
————— milk, 582
————— water, drinking, 583, 585
———— diuretics, 584
———— drugs, 584
————— coal-tars, 584
———— local, 583, 586
———— operations, 587
————— incision and drainage, 588
————— nephrectomy, 588
————— nephrotomy, 588
———— rest, 581
————— Gatch bed, 582
———— vaccines, 586
————— autogenous, 587
————— stock, 587

KIDNEY, diseases of, renal calculus, 606
——— diagnosis of, 606
———— ureteral catheterism, 606
———— x-ray, 606
——— forms of, 596
——— infection and, 607
——— references, 607
——— treatment, 606
———— surgical, 607
————— nephrectomy, 607
————— nephrolithotomy, 607
————— ureterolithotomy, 607
—— renal colic, 596
——— chill, 602
——— collapse, 602
———— treatment of, 602
————— cardiac stimulants, 602
——— fever, 602
——— nausea, 602
——— pain, 595, 596
———— relief of, 600
————— atropin, 601
————— chloral, 601
————— chloroform, 600, 601
————— cold, 599
————— douches, hot, 598
————— enteroclysis, 599
————— fomentations, 598
————— heat, 597
————— morphin, 600
————— posture, 600
————— poultices, hot, 598
————— warm drinks, 599
——— symptoms, 596, 597
——— treatment, 597
———— hydrotherapy, 598, 599
———— thermotherapy, 598, 599
———— urine suppression, 602
———— vomiting, 602
—— tuberculous kidney, 608
——— complications of, 614
——— diagnosis, 608, 609
———— cystoscopy in, 610
———— tubercle bacilli, 610
———— x-ray, 610
——— fever in, 613
——— gastro-intestinal disturbances, 614
———— anorexia, 614
———— constipation, 614
———— diarrhea, 614
——— hematuria, 613
———— treatment, 613
——— infection, 608
———— modes, 608
———— sites, 608
——— prophylaxis, 614
——— references, 614
——— symptoms, 609
———— frequent micturition, 609
———— hematuria, 609
———— pain, 609
———— polyuria, 609
——— treatment, 608
———— diet, 612
———— general, 611
———— hydrotherapy, 612
KIDNEY, diseases of, tuberculous kidney, treatment, surgical, 610
————— contra-indications, 610
————— indications, 610
————— mortality, 610
————— nephrectomy, 610
———— symptomatic, 613
———— tuberculin, 613
——— urine, 609
— physiology, normal, 508
——— internal secretion, 511
———— pressor substance, 511
————— Lewandowsky's experiment, 511
————— Pearce's experiment, 511
————— Tigerstedt's report, 511
——— renal secretion, 508
———— Bowman's theory of, 509
———— Cushny's theory, 509
———— Ludwig's theory, 508
——— urea, origin and significance, 510
——— urine composition, 510
—— pathological, 512
——— edema, 513
———— causative factors, 513
————— body fluids, increase in, 514, 515
————— chlorid retention, 513
————— nephritis, 514
————— vascular injury, 513, 514, 515
————— water retention, 513, 515
———— diuretics, 514
———— Fischer's theory, 516
———— nephritis and, 513, 515
———— uranium nephritis and, 514, 515
——— increased blood-pressure, 516
———— cardiac hypertrophy and, 517
——— nephrotoxic substances, 512
———— arsenic, 512, 524
———— chromium, 512, 524
———— cantharides, 512, 523, 552
———— mercury, bichlorid of, 512, 524, 552
———— diphtheria toxins, 512
———— potassium chromate, 512
———— snake venom, 512
———— uranium, 512
——— tests, 520
———— blood urea, determination of, 521
———— blood uric acid and creatinin, 522
———— diet, renal test, 522
———— non-protein nitrogen in blood, determination of, 522
———— phenolsulphonephthealein, 521
———— phthalein, 522
——— uremia, 518
———— causes, 518
————— nephrolysins, 519
————— renal elimination, imperfect, 519
———— symptoms, 518
—— *See also* Kidney, diseases of.
KIDNEY STONE. *See* Renal Calculus.

Kidneys, pathological physiology. *See* Kidney, diseases of.
Kingscote's breathing exercises, in pulmonary emphysema, 117
Klebs-Loeffler infection. *See* Larynx, diseases of, laryngitis
Koch's bacillus emulsion, in tuberculosis of larynx, 27
Krogh spirometer, in diagnosis of congenital heart disease, 355

Lacertililia, 892
Laryngismus strindulus. *See* Larynx, diseases of.
Laryngitis. *See* Larynx, diseases of.
Larynx, diseases of, 20
—— laryngitis, 20
——— acute, 20
———— treatment, 20
————— aconite root, 20
————— asperin, 20
————— calomel, 20
————— cold compresses, 20
————— children and, 22
————— croup tent, 20
————— Dobell's solution, 21
————— Dover's powder, 20
————— internal, 20
————— intralaryngeal applications, 22
————— prophylactic, 20
————— quinin, 20
————— Seiler's solution, 21
————— spray, 21
————— steam inhalers, 20
————— tartar emetic, 21
——— chronic, 22
———— adenoids and, 23
———— chorditis nodosa and, 22
———— gout in, 22
———— hyperplastic gland tissue in, 22
———— hypertrophic, 22
———— nasal stenosis in, 22
———— pachydermatous variety, 22
———— rheumatism in, 22
———— subglottic, 22
———— syphilis in, 22
———— tonsils and, 23
———— treatment, 22
————— alcohol forbidden, 22
————— astringents, 22
————— caustics, 22
————— climate, 22
————— potassium iodid, 22
————— tobacco forbidden, 22
——— chronic atrophic, 25
———— treatment, 25
————— hypodermics of green citrate of iron, 25
————— intratracheal injections, 25
————— iodid of potassium, 25
————— local applications, 25
————— phosphorus, 25
————— pilocarpin, 25
————— spray, 25
——— edematous, 23
———— systemic disease and, 23

Larynx, diseases of, laryngitis, edematous, treatment, 23
————— alkaline steam inhalations, 23
————— calomel, 23
————— codein, 23
————— Dover's powder, 23
————— hot astringent gargles, 23
————— intubation, 23
————— scarification, 23
————— tracheotomy, 23
——— membranous, 24
———— diagnosis, 24
———— treatment, 24
————— antitoxin, 24
————— calomel, 24
————— croup tent, 25
————— emetics in, 24
————— intubation, 25
————— spray, 24
————— tincture ferri chlor., 25
————— tracheotomy, 25
—— laryngitis, 20
——— spasmodic, 23
———— adenoids in, 23
———— children and, 23
———— tonsils hypertrophied in, 23
———— treatment, 23
————— alkaline steam inhalation,
————— antimony, 23
————— bromid of potassium, 24
————— bromid of sodium, 24
————— croup kettle, 24
————— ipecac, 23
————— of muscular spasm, 23
———— uvula, elongated, 23
—— syphilis of the upper respiratory tract, 30
——— saddle-nose in, 30
——— stenosis and, 30
——— trachea, gummata or ulceration,
——— treatment, 30
———— arsphenamin, 30
———— hypodermic medication, 30
———— iodin, 30
———— mercurials, 30
—— tuberculosis of the larynx, 25
——— anemia and, 28
——— heredity in, 25
——— syphilitic infection and, 28
——— treatment, 25
———— altitude, 26
———— antitoxins, 27
———— climate, 26
———— Deny's filtrate, 27
———— drugs, 27
———— fulguration, 29
———— hygienic, 26
———— injections, intralaryngeal, 29
———— Koch's bacillus emulsion, 27
———— prophylactic, 25
———— sera, 27
———— sunlight, direct in, 27
———— surgical, 29
———— tuberculin, 27
———— vaccines, 27
Lassar's paste, in eczema, 790

LATHYRISM, 901
Lavage, colonic, in phlebitis, 464
LEAD, as industrial poison, 667
Lead acetate in, hematuria, 504
— nephritis, 550
LEECHES. *See* Tropical diseases.
LEISHMANIASIS. *See* Tropical diseases, 858
Leriche's periarterial sympathectomy, in thrombo-angiitis obliterans, 478
Leukocytic extract in, bronchopneumonia, 104
— chronic pneumonia, 112
LICE, 895
Ligature, in thrombosis, 448
— of a patent ductus arteriosus, 358
LIGHT, in occupational diseases. *See* Industrial diseases.
LIGHTING, ARTIFICIAL, 730-732
LIGHTNING SHOCK, 747
Lindeman method of transfusion, 215
LIPOMA OF PLEURA. *See* Pleura, diseases of
LITHIASIS. *See* Kidney, diseases of, nephrolithiasis.
LIVER, FLUKES, 874
LOA INFECTION. *See* Tropical diseases, filariasis.
LOBAR PNEUMONIA, rare sequel of foreign body in bronchi, 80
Lobelia, in bronchitis, 45
LOCALIZED RUPTURE OF MUSCLES. *See* Rupture of muscles, localized.
LOUSE, in transmission of relapsing fever, 837
LOW COUNTRY MORNING DIARRHEA. *See* Tropical diseases.
LUDWIG'S THEORY of renal secretion, 508
Lumbar puncture, in uremia, 428
Luminal, in phlebitis, 464
LUNG ABSCESS. *See* Lungs, diseases of.
LUNG FLUKES, 877
LUNG TUMOR. *See* Lungs, diseases of.
LUNGS, diseases of, 89
— — abscess, 119
— — — bronchiectatic cavities and, 123
— — — etiological factors, 120
— — — — aspiration, 120
— — — — embolism, 120
— — — — pneumonia, 120
— — — gangrene and, 119
— — — glycosuria and, 125
— — — location, 121
— — — ruptured empyema mistaken for, 123
— — — symptoms, 122
— — — treatment, 123
— — — — diet, 124
— — — — medical, 123
— — — — — mortality, 125
— — — — — posture, 124
— — — — — strapping, 124
— — — — surgical, 124
— — — — — differential pressure apparatus, 124
— — — — — mortality in cases of, 125
— — — — — postoperative, 125
— — — — — results of, 125
LUNGS, diseases of, abscess, tumors and, 123
— — — x-ray in localization of, 123
— — bronchopneumonia, 101
— — — causes, 102
— — — — clothing, 102
— — — — infectious diseases, 102
— — — — overcrowding, 102
— — — influenza type, 101
— — — pathology, 101
— — — symptoms, 102
— — — treatment, 102
— — — — adenoids, 102
— — — — circulatory collapse, 108
— — — — — drugs, 108
— — — — complications, 109
— — — — convalescence, 110
— — — — cyanosis, 107
— — — — distention, 108
— — — — dyspnea, 107
— — — — fever, 106
— — — — hygiene, 105
— — — — — diet, 105
— — — — — mustard plasters, 106, 107
— — — — oxygen, methods of administering, 107
— — — — prophylactic, 102
— — — — — cold baths, 102
— — — — — diet, 102
— — — — — mouth hygiene, 103
— — — — — vaccination, 103
— — — — sinuses, 102
— — — — specific, 103
— — — — — Avery method for determining type of pneumonia, 103
— — — — — drugs, 104
— — — — — leukocytic extract, 104
— — — — — serum, 103
— — — — — types of reactions, 104
— — — — — vaccines, streptococcic, 105
— — — — tonsils, 102
— — chronic pneumonia, 110
— — — epidemics of influenzal pneumonia and, 111
— — — pathology, 110
— — — physical signs, 111
— — — symptoms, 111
— — — treatment, 112
— — — — active, 112
— — — — — drainage by posture, 112
— — — — — leukocytic extract, 112
— — — — — mustard plasters, 112
— — — — — thorocoplasty, 112
— — — — — vaccines, autogenous, 112
— — — — prophylactic, 112
— — circulatory disturbances, 89
— — — congestion, active, 91
— — — — — influenza and, 91
— — — — — irritating gases and, 91
— — — — — malaria and, 91
— — — — — pleurisy and, 91
— — — — — pneumonia and, 91
— — — — — pulmonary embolus and, 91
— — — — — scarlatina and, 91
— — — — — streptococcic conditions and, 91
— — — — — treatment, 91

LUNGS, diseases of, circulatory disturbances, congestion, active, tuberculosis and, 91
————— typhoid and, 91
———— hypostatic, 94
————— causes, 94
————— pneumonia and, 94
————— symptoms, 94
————— treatment, 94
———— passive, 92
————— arteriosclerosis and chronic, 93
————— bronchitis and chronic, 93
————— causes, 92
————— chronic, 93
————— hypertension and, 93
————— pathology, 93
————— pulmonary edema and, 93
————— symptoms, 93
————— treatment, 94
————— venesection, 94
——— edema, 95
———— etiology, 95
———— fibrosis of lung and, 95
———— influenza and, 95
———— insufficiency of cornary arteries and, 96
———— mechanical pressure of vagus and, 95
———— of gradual onset, 95
———— pathology, 96
———— pleurisy and, 95
———— pneumonia and, 95
———— rheumatism and, 95
———— scarlet fever and, 95
———— superacute, 95
———— symptoms, 96
———— treatment, 97
————— after, 97
————— amyl nitrite pearls, 97
————— aspiration of blood, 97
————— catheterization of lung, 97
————— diet, 97
————— digitalization, 97
————— drugs, 97
————— electrization over vagi, 97
————— hot packs, 97
————— preventive, 97
————— rest, 97
————— venesection, 97
———— tuberculosis and, 95
———— tumors, mediastinal or pulmonary and, 95
———— typhoid and, 95
——— embolism, 98
———— air emboli, 98
———— causes, 98
———— etiology, 98
———— fractures of long bones accompanied by, 98
———— large emboli, 99
————— symptoms, 99
————— treatment, 99
———— oil emboli, 99
———— small emboli, 99
————— causes, 99
————— prognosis, 100

LUNGS, diseases of, circulatory disturbances, embolism, small emboli, symptoms, 100
————— treatment, 100
——— factors causing, 90
———— pulmonary pressure, 89
————— increased, 90
————— lowered, 90
———— venous pressure, increased, 90
——— thrombosis, 98
—— emphysema, 113
——— chronic bronchitis and, 114
——— epidemic influenza pneumonia and, 113
——— Freund's theory and operation, 115
——— pathology, 113
——— prophylaxis of, 114
———— digitalis in, 114
——— treatment, 116
———— abdominal distention, correction of, 117
———— climate, 116
———— drugs, 118
———— hygiene, 116
———— mechanical devices, 117
————— Boghean apparatus, 117
————— Hofbauer's "expirator," 118
————— Kingscote's breathing exercises, 117
————— Rossbach chair, 117
————— Waldenburg's pneumatic cabinet, 118
———— occupation, 116
———— vaccinations, desensitizing, 117
—— gangrene, 119
——— fetid bronchitis and, 123
——— predisposing factors, 119
——— symptoms, 122
—— references, 129-133
—— tumors, 126
——— diagnosis, 126
———— X-ray in, 126, 128
——— treatment,
———— differential air pressure, 126
———— positive and negative pressure apparatus, 127
————— Brauer's apparatus, 127
————— Flint's apparatus, 127
————— Meltzer-Auer apparatus, Elsberg's modification of, 127
————— Robinson's apparatus, 127
————— Willy Meyer's apparatus, 127
LUNG-SUPPURATION. *See* Bronchi, foreign bodies in.
LYMPHOSARCOMA. *See* Mediastinum, diseases of, tumors.

Macewen's operation, in thoracic aneurysm, 445
Mackenzie spray, in laryngitis, 22
Male fern in, intestinal distomiasis, 876
— teniasis, 873
MALINGERING, 793
MALTA FEVER. *See* Tropical diseases, undulant fever.

MAMMALS, POISONOUS. *See* Poisons—reptiles—fish—anthropods and plants, affections due to.
MANGANESE, as industrial poison, 672
Martin's spring balance test, in industrial fatigue, 752
Massage, abdominal, in movable kidney, 503
Massage in, aneuresis, 503
— arteriosclerosis, 370, 375
— lead poisoning, 671
— muscular rheumatism, 488
— nephritis, 528
Matas' method of operating, in thoracic aneurysm, 445
Mechanical devices, in pulmonary emphysema, 117
Mechanical removal of fluid, in chronic myocarditis, 255
MEDIASTINAL TUMORS. *See* Mediastinum, diseases of.
MEDIASTINITIS. *See* Mediastinum, diseases of.
MEDIASTINOPERICARDITIS, INDURATIVE. *See* Mediastinum, diseases of.
MEDIASTINUM, diseases of, 172
— — mediastinitis, 177
— — — suppurative, 177
— — — — operations in the neck causing, 177
— — — — treatment, 177
— — — — — incision and evacuation, 177
— — — — — prevention of suppuration, 177
— — — — — trephining of, sternum, 177
— — mediastinopericarditis, indurative, 181
— — — treatment, 181
— — — — surgical, 181
— — — — — cardiolysis, 181
— — — — — results, 182
— — — valvular heart diseases and, 181
— — references, 182, 183
— — tuberculosis of the mediastinal lymph nodes, 178
— — — cause, 178
— — — diagnosis, 179
— — — prophylaxis, 178
— — — — adenoids, 178
— — — — diet, 178
— — — — enlarged tonsils, 178
— — — — fresh air, 178
— — — treatment, 179
— — — — climate, 179
— — — — diet, 179
— — — — drugs, 179
— — — — hygiene, 179
— — — — medical, 179
— — — — salt baths, 179
— — — — rest, 179
— — — — sea-bathing, 179
— — — — surgical methods, 179
— — — — symptomatic, 180
— — — — — bathing, 180
— — — — — drugs, 180
— — — — — dysphagia and, 180
— — — — — dyspnea and, 180
MEDIASTINUM, diseases of, tuberculosis, treatment, symptomatic, oxygen inhalation, 180
— — — — — tracheotomy, 180
— — — — tuberculin, 180
— — tumors, 172
— — — erysipelas and, 174
— — — lymphosarcoma amenable to palliation treatment, 174
— — — non-malignant, 173
— — — — dermoid cysts, 173
— — — — teratomata, 173
— — — Pott's disease and, 178
— — — treatment, 172
— — — — curative, 172
— — — — palliative, 174
— — — — — surgical intervention, 172
— — — — — drugs, 174, 176
— — — — — lymphosarcoma amenable to, 174
— — — — — operative procedures, 174, 176
— — — — — oxygen inhaled, 176
— — — — — radium, 176
— — — — — relief of dyspnea, 176
— — — — — tracheotomy, 176
— — — — — x-ray, 175
MEDINA WORM. *See* Tropical diseases, filariasis.
Meltzer-Auer air pressure apparatus, in lung tumors, 127
MEMBRANOUS LARYNGITIS. *See* Larynx, diseases of, 24
MENINGEAL HEMORRHAGE, in essential hypertension, 417
MERCURIAL NEPHRITIS. *See* Kidney, diseases of.
Mercurials, in syphilis of upper respiratory tract, 30
Mercury, acid nitrate of, cauterization in tuberculous cystitis, 619
MERCURY, as industrial poison, 6,2
Mercury, bichlorid, in ozena, 19
— — instillations, in tuberculous cystitis, 619
— **compounds** in endocarditis, 213
Mercury in, syphilitic aortitis, 440
— thoracic aneurysm, 443
— thrombo-angiitis obliterans, 475
MESENTERIC EMBOLISM, 458
MESENTERIC THROMBOSIS, 450
METABOLISM, in lithiasis, 604
METALS AND THEIR COMPOUNDS, as industrial poisons. *See* Industrial diseases.
METEORRHAGIA, in essential hypertension, 417
METHYL ALCOHOL, as industrial poison, 681
METHYL BROMID, as industrial poison, 688
Meyer's air pressure apparatus, in, of lung tumor, 127
Meyer's method of injections, in thrombo-angiitis obliterans, 477
MICROCOCCUS MELINTENSIS, causing undulant fever, 812
MICTURATION, FREQUENT, in tuberculous kidney, 609
Milk, in lead poisoning, 671

Milk diet in, hill diarrhea, 823
— sprue, 822
MINERS' NYSTAGMUS, 727
Monochloracetic acid, in chronic rhinitis, 7
MORPHIN, effect on pulmonary pressure of, 91
Morphin in, angina pectoris, 279
— bronchopneumonia, 109
— chronic myocarditis, 236, 251
— embolism, 455, 457
— essential hypertension, 393, 394, 395, 399, 400, 404, 413, 416, 428
— large pulmonary emboli, 99
— pericarditis, 197
— kidney of pregnancy, 578
— lead poisoning, 671
— muscular rheumatism, 488
— myositis, 484
— nephritis, 549, 550
— phlebitis, 464
— pulmonary edema, 97
— pulmonary embolism, 101
— pyelitis, 584
— renal colic, 600
— renal tuberculosis, 613
— thoracic aneurysm, 444
— thrombo-angiitis obliterans, 475
MOSQUITOES, in transmission of yellow fever, 845, 846
MOTHS, POISONING BY, 497
MOUTH BREATING, AVOIDANCE OF, in chronic bronchitis, 49
MOVABLE KIDNEY. *See* Kidney, movable.
Murphy drip in, anuresis, 503
— pyelitis, 585
MUSCLES, diseases of, 483
—— cramps, 489
——— treatment, 489
—— myositis, 483
——— fibrosa, 486
———— treatment of, 486
——— ossificans, 486
———— treatment, 487
————— operative, 487
———— types, 486
——— polymyositis, acute, 483
———— diagnosis, 484
———— nature, 483
———— prognosis, 484
———— symptoms, 483
———— treatment, 484
————— drugs, 484
————— electricity, 484
————— gymnastics, 484
————— hot, dry packs, 484
————— massage, 484
——— primary acute suppurative, 485
———— clinical picture, 485
———— treatment, 485
——— secondary suppurative, 486
——— syphilis of muscles, 484
———— forms, 484
——— tuberculous, 486
———— treatment, 486
————— incision of broken down nodules, 486
MUSCLES, diseases of, myositis, tuberculous, treatment, x-ray, 486
—— references, 490, 491
—— rheumatism, 487
——— factors of etiological importance, 487
———— chilling, 487
———— circulatory disturbances, 487
———— focal infection, 487
———— toxins, 487
———— trauma, 487
———— true rheumatism, 487
——— prevention, 488
——— treatment, 488
———— acupuncture, 488
————— technic, 489
———— bowels, evacuation of, 488
———— diet, 488
———— drugs, 488
———— electricity, 488
———— massage, 488
———— rest, 488
———— saline solution, injection of, 488
———— warmth, 488
———— water drinking, 488
—— rupture of muscles, localized, 489
——— treatment, 490
MUSCULAR CRAMPS. *See* Muscles, diseases of.
MUSCULAR RHEUMATISM. *See* Muscles, diseases of.
Mustard plasters in, angina pectoris, 279
— bronchopneumonia, 106, 107
— chronic pneumonia, 112
— congestion of lungs, 92, 94
— pulmonary embolism, 100
MYALGIA. *See* Muscles, diseases of.
MYIASIS, 897
MYOCARDITIS, 227
— *See also* Heart, diseases of.
MYOSITIS. *See* Muscles, diseases of.
MYOSITIS FIBROSA. *See* Muscles, diseases of.
MYOSITIS OSSIFICANS. *See* Muscles, diseases of.
MYXOMA OF BLADDER. *See* Bladder, diseases of.

NAPHTHA, as industrial poison, 675
NAPHTHALENE, as industrial poison. *See* Anilin.
NAPHTHYLAMINS, as industrial poisons. *See* Anilin.
NARCOLEPSY. *See* Trypanosomiasis.
Nasal irrigation, in acute nasal sinusitis, 11
NASAL POLYPI, in acute bronchitis, 34
NAUSEA, in renal colic, 602
NEGRO LETHARGY. *See* Trypanosomiasis.
NELAVAN. *See* Trypanosomiasis.
NEMATODE INFECTION. *See* Tropical diseases.
Neo-arsphenamin, in Oroya fever, 830
Neokharsivan, in Trypanosomiasis, 851
Nephrectomy in, hydronephrosis, 594
— movable kidney, 503

Nephrectomy in, renal tuberculosis, 610
NEPHRITIS. *See* Kidney, diseases of.
NEPHROLITHIASIS. *See* Kidney, diseases of.
Nephrolithotomy, in renal calculus, 607
Nephrotomy in, anuresis, 504
— hematuria, 504
— hydronephrosis, 594
— pyelitis, 588
NEURITIS, caused by cold, 715
NEUROCIRCULATORY ASTHENIA. *See* Irritable heart.
NEUROSES, CARDIAC, 312.
—— *See also* Heart, diseases of.
NEUROSES, OCCUPATIONAL, 766
—— *See also* Industrial diseases.
NEUROSES, OF BLADDER. *See* Bladder, diseases of.
NICOTIN, as industrial poison, 688
NITRIC ACID, as industrial poison, 687
Nitrite of amyl in, angina pectoris, 278
— arrow poisoning, 902
— arteriosclerosis, 372
— essential hypertension, 400
— hemoglobinuria, 507
— pulmonary edema, 97
NITRITES, effect on pulmonary pressure, 91
Nitrites in, angina pectoris, 278, 282
— arteriosclerosis, 372
— thoracic aneurysm, 444
— thrombo-angiitis obliterans, 475
NITROBENZOL, as industrial poison, 681
NITROGEN, NON-PROTEIN, in blood, test for determination of, in nephritis, 522
Nitrogen gas infection, in bronchiectasis, 64
NITROGLYCERIN, as industrial poison, 682
— effect on pulmonary pressure of, 91
Nitroglycerin in, angina pectoris, 278
— arteriosclerosis, 372
— essential hypertension, 394, 400, 412
— myocarditis, 251
— nephritis, 542
— pulmonary edema, 97
— thrombo-angiitis obliterans, 475
— vascular accidents, 413
Nitrous ether, spirits of, in arrow poisoning, 902
NON-METALS AND THEIR COMPOUNDS, as industrial intoxications, 674
NON-OBLITERATIVE PHLEBITIS, 461
NOSE, diseases of, 3
—— epistaxis, 17
——— atheromatous subjects and, 17
——— causal factors, 17
——— high blood-pressure attending, 18
———— postnatal ulceration in, 18
———— postoperative, treatment, 18
——— recurrent, 18
———— pulmonary hemorrhage, 18
———— tumor, 18
——— treatment, 17
———— antitoxin, 17
———— astringents, powerful, 18
———— cauteries, 18
———— chlorid of iron, 18
———— hydrastinin hydrochlorid, 18
———— internal medication, 18
NOSE, diseases of, epistaxis, treatment, stypticin, 18
———— tampons, nasal, 18
—— ozena, 19
——— treatment, 19
———— alkaline solutions, antiseptic, 19
———— antidiphtheritic serum, 19
———— bichlorid of mercury spray, 19
———— creolin, 19
———— electricity, 19
———— ichthyol, 19
———— internal medication, 19
———— iodin, 19
———— massage, 19
———— physical measures, 19
———— vaccine, 19
—— rhinitis, 4
——— acute, 4
———— causes, 6
———— prophylaxis, 4
———— recurrent, 4
———— treatment, 5
————— cauterization, 5
————— drugs, 5
————— spray, 5
————— vaccines, 5
——— atrophic, 8
———— climate, 8
———— remedies, 8
———— treatment, 8
————— cleansing measures, 9
————— drugs, 8
————— electricity, 9
————— iodin, 8
————— oleaginous preparations, 9
————— thyroid extract, 8
——— chronic, 6
———— etiology, 6
———— prophylaxis, 6
———— treatment, 7
————— drugs, 6
————— electrolysis, 7
————— monochloracetic acid in, 7
————— trichloracetic acid in, 7
——— membranous, 7
———— antitoxin, 7
———— prophylaxis, 7
———— treatment, 7
————— drugs, 7
—— rhinoscleroma, 4
——— treatment, 10
———— Schroetter's tubes, 11
———— surgical methods, 11
———— vaccine, autogenous, 10
—— sinusitis, 11
——— hyperplastic ethmoiditis, 16
———— asthma in, 17
———— causes, 16
———— influenza producing, 16
———— treatment, 16
——— nasal, 11
———— acute, 11
————— diagnosis, 11
————— grip and, 11
————— nasal catarrh and, 11
————— nasal irrigation, 11

NOSE, diseases of, sinusitis, nasal, acute, operation, 12
— — — — — spray, 12
— — — — — treatment, 11
— — — — chronic, 13
— — — — — detection, 13
— — — — — headache, 13
— — — — — optic nerve involvement, 13
— — — — — systemic infection and, 13
— — — — — tooth abscess in, 14
— — — — — treatment, 14
NOSEBLEED. *See* Epistaxis.
Novacain, as anesthetic in bladder tumor, 636
Nutrition, in prevention of bronchopneumonia, 102
Nux vomica, in enuresis, 631
NYSTAGMUS, miners', 727

OBLITERATING ENDARTERITIS, 375
OBLITERATIVE PHLEBITIS, 461
OCCUPATION, influence of, on pulmonary emphysema, 116
OCCUPATIONAL DERMATOSES. *See* Industrial diseases.
OCCUPATIONAL DISEASES. *See* Industrial diseases.
OCCUPATIONAL NEUROSES. *See* Industrial diseases.
OCULAR FATIGUE, 725
ŒSOPHAGOSTOMIASIS. *See* Tropical diseases, nematode infection.
Oleaginous preparations, in atrophic rhinitis, 9
OLIGURESIS, in lithiasis, 605
OPHTHALMIA, ELECTRIC, 720, 747
Opiates, in pleurisy, 145
Opium, in bronchitis, 42
Optic nerve involvement in chronic nasal sinusitis, 13
ORCHITIS, in trypanosomiasis, 849
ORIENTAL SORE. *See* Leishmaniasis, dermal.
OROYA FEVER. *See* Tropical diseases.
Orpiment, in prophylaxis of trypanosomiasis, 855
OSTEITIS DEFORMANS. *See* Bones, diseases of.
OSTEOMALACIA. *See* Bones, diseases of.
OSTEOARTHROPATHY, SECONDARY HYPERTROPHIC. *See* Bones, diseases of.
Ouabain, in myocarditis, 224
Oudin current in, bladder tumor, 636
— tuberculous cystitis, 619
Outdoor life, after thoracentesis, 162
OVERCROWDING, as cause of bronchopneumonia, 102
Oxygen, intravascular administration, in congenital heart disease, 346
— intravenous and intra-arterial administration, in congenital heart disease, 358
Oxygen gas, in pleurisy, 161
Oxygen in, bronchopneumonia, 107
— caisson-disease, 703, 706
Oxygen inhalation in, anilin poisoning, 675
— bronchiectasis, 65
Oxygen inhalation in, bronchitis, 45
— congenital heart disease, 343
— essential hypertension, 395
— industrial intoxications, 677, 682
— kidney of pregnancy, 578
— mediastinal tumors, 176
— methemoglobinuria, 506
— thrombosis, 449
OZENA. *See* Nose, diseases of.

Packs, ice-cold, in insulation, 810
Packs, hot, in anuresis, 503
— — myositis, 484
— — osteomalacia, 496
— — pulmonary edema, 97
— — renal colic, 598
PAGET'S DISEASE. *See* Osteitis deformans.
PAIN in, caisson-disease, 694
— tuberculous kidney, 609
Pancreatic extract, in sprue, 822
PAPILLOMA OF BLADDER. *See* Bladder, diseases of.
PAPPATACI FEVER. *See* Tropical diseases.
Paquelin cautery, in pericarditis, 194
Paracentesis, in pericarditis, 198, 202
Paraldehyd in, bronchitis, 43
— phlebitis, 464
PARONYCHIA. *See* Industrial diseases, 793
PAROXYSMAL DYSPNEA, 393
PAROXYSMAL TACHYCARDIA, 410
PASSIVE CONGESTION OF LUNGS. *See* Lungs, diseases of.
PERICARDITIS, 187
— *See also* Heart, diseases of.
PERICARDITIS, ACUTE, 188
PERINEPHRITIS. *See* Pyelitis.
PERIPHERAL THROMBOSIS. *See* Thrombosis; Phlebitis.
Permanganate of potassium, in tropical intoxications, 904
Phenacetin in, diseases of muscles, 484
— insulation, 811
— phlebitis, 464
Phenol, in endocarditis, 213
Phenosulphonephthalein, in functional kidney tests, 521
Phenolsulphonephthalein tests, in pyelitis, 583
PHLEBITIS, NON-OBLITERATIVE, 461
—obliterative, 461
— recurrent, 462
— *See* Blood vessels, diseases of.
PHLEGMASIA ALBA DOLENS. *See* Phlebitis, chronic.
PHLEBOTOMUS FEVER. *See* Pappataci fever.
PHOSGINE, as industrial poison, 687
PHOSPHORATED HYDROGEN, as industrial poison, 683
PHOSPHORUS, as industrial poison, 683
Phosphorus, in chronic atrophic laryngitis, 25
PHOTOPHOBIA, in electric ophthalmia, 721
— ultraviolet rays causing, 722
Phthalein test, in nephritis, 522
Picric acid, in industrial infections, 659

Pilocarpin in, bronchitis, 42, 61
— chronic atrophic laryngitis, 25
PITUITRIN, effect on pulmonary pressure of, 90
Pituitrin in, bronchopneumonia, 108
— hematuria, 504
PLANTS, AFFECTIONS DUE TO POISONOUS, *See* Poisons—reptiles—fish—arthropods and plants, affections due to.
PLASTIC BRONCHITIS. *See* Bronchi, diseases of.
PLEURA, diseases of, 134
—— pleural effusions, 134
——— diagnosis, 134
——— treatment, 134
———— thoracentesis in, 134
—— pleurisy, 136
——— absorption of exudates, 136
———— cause of death from, 140
———— fibrin, 137
———— fluid, 137
———— gases, 138
———— influence on circulation, 139
———— intrathoracic pressure, 139
———— mechanical effect, 139
——— carcinoma and, 142
——— clinical course, 142
——— emphyema and, 143
——— end result after absorption in, 143
——— fibrinous, 144
———— acute, 145
————— calomel, 145
————— hot and cold applications, 145
————— opiates, 145
————— strapping with adhesive plaster as, 145
————— symptoms, 145
————— treatment, 145
———— chylothorax, 163
————— treatment, 163
———— diagnosis, 144
———— hemorrhagic pleurisy, 163
———— hemothorax, 163
————— traumatic form of, 163
———— pneumothorax, 164
————— accidental, during thoracentesis, 164
————— artificial, for therapeutic purposes, 166
————— acutissimus,
————— emphysema and, 165
————— serous effusion and, 165
————— spontaneous, 165
————— traumatic, 165
————— tuberculous, 165
———— serofibrinous, 145
———— treatment, 145
————— adrenalin in, 160
————— after operation, 162
————— artificial respiration in, 160
————— aspiration method in operation, 154
————— atropin in, 160
————— autoserotherapy in, 161
————— baths, cold, 146
————— cathartics, 147
PLEURA, diseases of, pleurisy, fibrinous, treatment, caffein, 148
————— cardiovascular stimulants, 148
————— chronic serous effusion, 162
————— codein, 146
————— cold applications, 146
————— cough, 146
————— diaphoretics, 148
————— diet, 147
————— diuretics, 148
————— dry cupping, 146
————— exploratory puncture before operation, 154
————— exudates, role of, 147
————— fever, 146
————— hydrotherapy, 146
————— instrument used in operation, 153, 160
————— intrapleural injections, 160
————— iodids, 148
————— iodin, painting with, 146
————— location of puncture, 151
————— morphin, 146
————— operations contra-indicated, 151
————— operative measures, 149
————— oxygen gas, 161
————— position during operation, 152
————— preparation for operation, 152
————— quinin, 146
————— salicylates, 146
————— symptomatic, 146
————— thiosinamin, 161
————— thoracentesis, 149
————— thoracotomy, 162
————— transudates, rôle of, 147
————— trocar, in thoracentesis, 153 160
——— following tonsillar infection, 143
——— mediastinum, permeability of, 138
——— onset, 136
——— pain sense, 140
———— distribution of, 141
——— pathology, clinical, 136
——— rheumatic arthritis and, 143
——— treatment, 143
———— eradication of focus of infection, 143
———— fresh air, 144
———— iodids, 143
———— prophylactic, 143
———— rest, 143
———— salicylates in rheumatic forms, 143
—— references, 167-171
—— tumors of pleura, 166
——— carcinoma, 166
———— endothelial, 167
——— echinococcus cysts, 167
——— enchondroma, 166
——— fibroma, 166
——— lipoma, 166
——— sarcoma, 166
——— treatment, 167

PLEURA, TUMORS OF. *See* Pleura, diseases of.
PLEURISY. *See* Pleura, diseases of.
PLEURISY, FIBRINOUS. *See* Pleura, diseases of.
PLEURITIS. *See* Pleura, diseases of.
PLUMBISM. *See* Lead as industrial poison.
PNEUMOCONIOSIS. *See* Industrial diseases.
PNEUMONIA, CHRONIC. *See* Lungs, diseases of.
PNEUMOPERICARDIUM, 204
PNEUMOTHORAX. *See* Pleura, diseases of.
POISONOUS FISH, 893
POISONOUS MAMMALS, 894
POISONS, INDUSTRIAL. *See* Industrial diseases.
POISONS — REPTILES—FISH — ARTHROPODS AND PLANTS, affections due to, 890
— — arrow poisoning, 901
— — — varieties, 901
— — — — erythrophlœum, 902
— — — — — treatment, 903
— — — — strophanthus, 902
— — — — — treatment, 902
— — — — strychnos, 901
— — — — — treatment, 902
— — arthropods, 894
— — — caterpillars—butterflies — moths, 897
— — — — treatment, 897
— — — centipede bite, 895
— — — — symptoms, 895
— — — — treatment, 895
— — — flies and fleas, 897
— — — — treatment, 897
— — — insects, 896
— — — — treatment, 896
— — — jigger, 900
— — — — prophylaxis, 900
— — — — treatment, 900
— — — louse and bug poisoning, 896
— — — — treatment, 896
— — — myiasis, 897
— — — — aural, 898
— — — — dermal, 898
— — — — intestinal, 898
— — — — ocular, 898
— — — — rhinal, 898
— — — — varieties of flies causing, 897
— — — — treatment, 898
— — — spider bite, 894
— — — — effects, 894
— — — — treatment, 894
— — — tick poisoning, 895
— — — — effects, 896
— — — — treatment, 896
— — — — — preventive, 896
— — atriplicism, 900
— — — cause, 900
— — — symptoms, 901
— — — treatment, 901
— — fish, 893, 906
— — — preventive measures, 894
— — — symptoms, 893, 906
— — — treatment, 894, 906
— — lathyrism, 901
— — — treatment, 901
POISONS, lathyrism, mammals, 894
— — plants, 905
— — — varieties, 905
— — — — ackee, 905
— — — — — prophylaxis, 905
— — — — — treatment, 906
— — — — onyalai, 906
— — — — — treatment, 906
— — snake poisoning, 890
— — — colubine, 891
— — — — symptoms, 891
— — — prophylaxis, 892
— — — scorpion bite, 894
— — — — symptoms, 895
— — — — treatment, 895
— — — — — serum, 895
— — — treatment, 891
— — — — non-specific, 891
— — — — — artificial respiration, 892
— — — — — gastric lavage, 892
— — — — — potassium permanganate, 892
— — — — — strychnin, 892
— — — — specific, 891
— — — — — sera, 891
— — — venom, toxidity of, 890
— — — viperine, 891
— — — — symptoms, 891
— — tropical intoxications, 904
— — — treatment, 904
— — vertebrates other than snakes, 892
— — — batrachia, 892
— — — — salamanders, 892
— — — — toads, 892
— — — lacertilia, 892
— — — — Holoderma, 892
— — — treatment, 893
POLYURIA, in tuberculous kidney, 609
POLYPAPILLOMA. *See* Frambesia tropica.
POLYSEROSITIS, in adhesive pericarditis, 204
Potassium, salts of, in enuresis, 627
Potassium bichromate, in bronchitis, 47
Potassium iodid in, aortic or thoracic aneurysm, 443
— bronchitis, 47
— chronic laryngitis, 22
— phlebitis, 465
— syphilitic aortitis, 440
— thrombo-angiitis obliterans, 475
Potassium permananate in, acute cystitis, 618
— arrow poisoning, 902, 903
— snake poisoning, 892
Poultices, hot, in renal colic, 598
PREMATURE CONTRACTIONS OF HEART, 295
— *See also* Heart, diseases of.
PRESENILE SPONTANEOUS GANGRENE. *See* Thrombo-angiitis obliterans.
PRIMARY ACUTE SUPPURATIVE MYOSITIS. *See* Muscles, diseases of.
PROTEIN IN FOODS, table of, 536
PRUSSIC ACID, as industrial poison, 687
PSEUDO-ANGINA PECTORIS, 284
PSEUDOTRICHINOSIS. *See* Muscles, diseases of, polymyositis, acute

Psychotherapy, in irritable heart, 318
PULMONARY EDEMA. *See* Edema, pulmonary.
PULMONARY EMBOLISM. *See* Embolism, pulmonary.
PULMONARY EMPHYSEMA. *See* Emphysema, pulmonary.
PULMONARY THROMBOSIS. *See* Thrombosis, pulmonary.
PULSUS ALTERNANS, 310
— *See also* Heart, diseases of.
PUS INFECTIONS. *See* Industrial diseases.
PYELETIS. *See* Kidney, diseases of.
PYELOGRAPHY, in hydronephrosis, 593, 594
PYELONEPHRITIS. *See* Pyelitis.
PYOGENIC INFECTIONS, in cystitis, 617
PYOPERICARDIUM, 203

QUARTZ LAMP, 723
Quinidin sulphate in, chronic myocarditis, 262, 264, 265
— disorders of heart beat, 300, 305
— myocarditis, 225
Quinidin therapy, in auricular fibrillation, 305
— thrombosis, 449
Quinin and urea hydrochlorid, in bronchopneumonia, 104
Quinin enemata in, intestinal schistosomiasis, 879
— ulcerative colitis, 867
Quinin in, acute bronchitis, 36
— acute laryngitis, 20
— endocarditis, 213
— hematochyluria, 881
— hemoglobinuria, 507
— pleurisy, 146

RADIATION, and occupational diseases. *See* Industrial diseases.
Radiation, in bladder tumor, 643
RADIUM BURNS. *See* Burns.
RADIUM DERMATITIS, 737
Radium implantations, in bladder tumor, 650
Radium therapy in, arteriosclerosis, 371
— epithelioma, 791
— mediastinal tumors, 175
— x-ray dermatitis, 740
Radium. *See also* Industrial diseases, radiation, 735
Recompression, in caisson-disease, 704-706
RECURRENT PHLEBITIS, 462
Reëducation, in lead poisoning, 671
RELAPSING FEVERS. *See* Tropical diseases.
RENAL CALCULUS. *See* Kidney, diseases of, nephrolithiasis.
RENAL COLIC. *See* Kidney, diseases of, nephrolithiasis.
RENAL EMBOLISM, 458
RENAL LESIONS, in essential hypertension, 418
— — *See also* Blood vessels, diseases of.
RENAL SCLEROSIS, 374
— *See also* Blood vessels, diseases of.
RENAL SECRETION, theories of, 508
RENAL TEST DIET, in nephritis, 522
RENAL TUBERCULOSIS. *See* Kidney, diseases of, tuberculous.
REPTILES, AFFECTIONS DUE TO POISONOUS. *See* Poisons—reptiles—fish—arthropods and plants, affections due to.
Resection, in bladder tumor, 650
Rest in, acute coronary obstruction, 287
— amyloid disease of kidney, 576
— aortic or thoracic aneurysm, 442, 443
— arteriosclerosis, 371
— benzene poisoning, 677
— embolism, 457
— essential hypertension, 382, 385, 386, 404, 408, 412, 423, 426
— muscular rheumatism, 488
— myocarditis, 224, 235
— nephritis, 527, 542, 552, 565
— osteomalacia, 495
— passive congestion of lungs, 94
— pulmonary edema, 97
— pulmonary emboli, 99, 100
— pyelitis, 582
— sprue, 822
— syphilitic aortitis, 440
— thrombosis, 449
RHINITIS, acute. *See* Nose, diseases of.
— atrophic. *See* Nose, diseases of.
— chronic. *See* Nose, diseases of.
— membranous. *See* Nose, diseases of.
RHINOSCLEROMA. *See* Nose, diseases of.
Rhus toxicodendron, in enuresis, 620
Ringer's solution, in thrombo-angiitis obliterans, 477
RINGWORM. *See* Tinea.
Robinson's air pressure apparatus, in lung tumors, 127
Roentgen ray. *See* X-ray.
Rossbach chair, in pulmonary emphysema, 117
RUPTURE OF MUSCLES, localized. *See* Muscles, diseases of.
Ryan's reaction, in industrial fatigue, 753

SADDLE-NOSE, in syphilis of upper respiratory tract, 31
Sahli's lavage of blood, in methemoglobinuria, 506
SALAMANDER POISONING, 892
Salicylates in, endocarditis, 213
— insulation, 811
— muscular rheumatism, 488
— myositis, 484
— pericarditis, 195
—rheumatic forms of pleurisy, 143, 146
Saline cathartics, in nephritis, 529, 553
Saline infusions, in anuresis, 503, 504
Saline solution, local injection of, in muscular rheumatism, 488
— normal, in methemoglobinuria, 506
Salipyrin, in myositis, 484
Salol, in distomiasis, 875
SALT, amount of, in average portion of food, 533
Salvarsan. *See* Arsphenamin.
SAND-FLY FEVER. *See* Pappataci fever.

Sarcoma of bladder, 633

Sarcoma of pleura, 160

Scarlet red, in ulcer, 796

Schistosomiasis. *See* Tropical diseases.

Schneider's cardiovascular rating, in industrial fatigue, 753, 754

Schroetter's tubes in, rhinoscleroma, 11

— syphilis of upper respiratory tract, 31

Scorpion bites, 894

Sea bathing, in tuberculosis of mediastinal lymph nodes, 179

Secondary hypertrophic osteoarthropathy. *See* Osteoarthropathy, secondary hypertrophic.

Secondary suppurative myositis, 486

Sedatives, in essential hypertension, 382, 398, 412, 413, 426

Seiler's solution, in laryngitis, 21

Senile arteriosclerosis, 376

Serofibrinous pleurisy, 145

Serum sickness, as reaction to serum treatment for bronchopneumonia, 104

Serum therapy, in anthrax, 786

— bronchitis, 53

— bronchopneumonia, 103

— endocarditis, 212, 221

— scorpion bite, 895

— snake poisoning, 891

— tuberculosis of the larynx, 27

— undulant fever, 813

— yellow fever, 844

Shaeffer method of artificial respiration, in carbon dioxid poisoning, 680

Shock, electrical, 743

Siderosis. *See* Industrial diseases, pneumoconiosis.

Silicosis. *See* Industrial diseases, pneumoconiosis.

Silver nitrate, in enuresis, 627

Silver nitrate irrigations, in acute cystitis, 618

— endemic hematuria, 879

Singer's nodes, and chronic laryngitis, 22

Sinus arhythmia, 293

Sinus bradycardia, 294

Sinus tachycardia, 294

Sinuses, accessory, diseases of, 3

— diseased, and bronchopneumonia, 102

Sinusitis, acute nasal. *See* Nose, diseases of.

Siriasis. *See* Insolation.

Sleep in, arteriosclerosis, 371

— essential hypertension, 408, 423

Sleeping sickness. *See* Trypanosomiasis.

Snake poisoning. *See* Poisons—reptiles—fish—arthropods and plants, affections due to.

Sodium, salicylates of, in pyelitis, 585

Sodium antimonyl tartrate, in visceral leishmaniasis, 861

Sodium bromid, in aortic or thoracic aneurysm, 443

Sodium carbonate, in insolation, 810

Sodium citrate, in thrombo-angiitis obliterans, 477

Sodium chlorid content of various foods, 533

Sodium chlorid in, insolation, 810

— nephritis, 531, 556, 560

Sodium hypochlorid enemata, in intestinal schistosomiasis, 879

Sodium iodid, in aortic or thoracic aneurysm, 443

Sodium nitrite, in nephritis, 542

Sodium theobromin salicylate, in angina pectoris, 283

Soldier's heart, 313

Soporifics, in bronchitis, 43

Sparking, in bladder tumor, 636

Spasm, in occupational neuroses, 778

Spasmodic laryngitis. *See* Larynx, diseases of.

Spider bite, 894

Spirochetoses. *See* Tropical diseases.

Spleen, enlargement in, relapsing fever, 837

— trypanosomiasis, 849

Splenic embolism, 458

Spontan-gangrän. *See* Thrombo-angiitis obliterans.

Spray in, bronchitis, 40, 55, 56, 59

— laryngitis, 21, 22, 25, 28, 31

— rhinitis, 5, 7, 8, 9

— sinusitis, 12

Sprue. *See* Tropical diseases.

Steam inhalers, in acute laryngitis, 20

Steele's technic of infections, in thrombo-angiitis obliterans, 477

Stegomyia calopus, in transmission of yellow fever, 845, 846

Sterilized air, after removal of pericardial effusion in hydropericardium, 202

Sterility, radium causing, 737

— x-rays causing, 737

Stimulants, heart, in anuresis, 503, 504

Stimulants, in pericarditis, 196

Stokes-Adams' syndrone, in heart-block, 309

Strapping with adhesive plaster, in pleurisy, 145

Strongyloides stercoralis, 884

Strongylosis. *See* Tropical diseases, nematode infection.

Strophanthin in, bronchopneumonia, 109

— embolism, 457

— myocarditis, 224

— pericarditis, 197

Strychnin in, arrow poisoning, 902

— bronchitis, 45, 55, 60

— bronchopneumonia, 109

— embolism, 457

— paralysis of bladder, 623

— pulmonary edema, 97

— snake poisoning, 892

Strychnin sulphate, in enurisis, 631

Strychnos, as arrow poisoning, 901

Styptisin, in epistaxis, 18

Subacute endocarditis, 207

— *See also* Heart, diseases of.

Sulphocarbolates, in endocarditis, 213

Sulphonal, in bronchitis, 43

Sulphur dioxid, as industrial poison, 687

Sulphurated hydrogen, as industrial poison, 683

SULPHURIC ACID, as industrial poison, 687
Sunlight in, phlebitis, 467
— rickets, 722
SUNSTROKE. *See* Insolation.
Suprapubic cystotomy, in bladder tumor, 638
Sweating in, anuresis, 503
— kidney of pregnancy, 577
— nephritis, 533, 542, 548, 551, 556, 563
SYPHILITIC AORTITIS. *See* Aortitis, syphlitic.
SYPHILIS, in etiology of chronic myocarditis, 232
SYPHILIS OF MUSCLES. *See* Muscles, diseases of.
SYPHILIS OF UPPER RESPIRATORY TRACT. *See* Larynx, diseases of.

TACHYCARDIA, PAROXYSMAL, 297
— — *See also* Heart, diseases of.
Tampon, nasal, in epistaxis, 18
Tannic acid in, arrow poisoning, 902
— hematuria, 504
TAPEWORMS. *See* Teniasis.
Tapping. *See* Thoracentesis.
Tartar emetic in, acute laryngitis, 21
— dermal leishmaniasis, 865
— trypanosomiasis, 850, 851
— ulcerating granuloma of pudenda, 831
— visceral leishmaniasis, 860
TELLURIUM, as industrial poison, 689
TENIASIS. *See* Tropical diseases.
TERMINAL INFECTION, in essential hypertension, 429
— *See also* Blood vessels, diseases of.
Tests, agglutination, in diagnosis of undulant fever, 813
—functional kidney, in nephritis, 520
— in diagnosis of pyelitis, 581, 583, 588
TETRACHLORETHANE, as industrial poison, 684
TETRALIN. *See* Tetrachlorethane.
Thallin sulphate instillations, in tuberculous cystitis, 619
Theobromin in, arteriosclerosis, 372
— in pulmonary edema, 97
Theobromin-sodiosalycylate, in nephritis, 545
Theocin, in nephritis, 545
THERMAL REACTION, to serum treatment for bronchopneumonia, 104
THERMIC FEVER. *See* Insolation.
Thermotherapy in, muscular rheumatism, 488
Thermotherapy in, phlebitis, 646
— renal colic, 597
— thrombo-angiitis obliterans, 475
Thiosinamin in, pleurisy, 161
— thrombo-angiitis obliterans, 475
Thoracentesis in, cardiac failure with edema, 409
— pleurisy, 149
THORACIC ANEURYSM, 443
— *See also* Blood vessels, diseases of.
THORACOTOMY, in pleurisy, 162
THREE-DAY FEVER. *See* Pappataci fever.
THROMBO-ANGIITIS OBLITERANS, 469
— *See also* Blood vessels, diseases of.
THROMBOSIS, 466
— See also Blood vessels, diseases of.
— pulmonary; Lungs, diseases of.
Thymol in, intestinal distomiasis, 876
— œsophagostomiasis, 888
Thyroid extract in, atrophic rhinitis, 8
— enuresis, 620, 629
— heart-block, 309
TICK, in transmission of relapsing fever 838
TICKS, POISONING by, 895
TIGHT LACING, as cause of movable kidney, 502
TINEA. *See* Industrial diseases.
TNT, as industrial poison. *See* Trinitrotoluene.
TOADS, POISONING by, 892
TOBACCO, effects of, in chronic bronchitis, 50
TOCHIL. *See* Endemic hemoptysis.
TOLUENE, as industrial poison. *See* Anilin.
TOLUINDIN, as industrial poison. *See* Anilin.
Tonics, in occupational neuroses, 781
Tonsillectomy in, congenital heart disease, 357
— pleurisy, 143
TONSILS, acute bronchitis and, 34
— bronchopneumonia and, 102
— chronic laryngitis and, 23
— chronic myocarditis and, 232
— chronic rhinitis and, 6
— pericarditis and, 189
TOXIC TYPE OF ARTERIOSCLEROSIS, 377
Tracheotomy, in mediastinal tumors, 176
Transfusion, blood. *See* Blood transfusion.
— salt, in nephritis, 558
TREMATODE INFECTION. *See* Tropical diseases.
TREMOR, in occupational neuroses, 778
TRENCH FOOT, 715
Trendelenburg position, in bronchiectasis, 63
Trephining of sternum, in mediastinitis, 177
Trichloracetic acid in, chronic rhinitis, 7
— epistaxis, 18
TRICHOCEPHALIASIS. *See* Tropical diseases, nematode infection.
TRICHURIS TRICHIURA, 886
TRINITROBENZOL. *See* Nitrobenzol.
TRINITROTOLUENE, as industrial poison, 685
TRIONAL in, bronchitis, 44
— insolation, 811
Trocar, in thoracentesis, 153, 160, 200
TROPICAL DISEASES, 799
— cestode infection, 870
— distomiasis, 874
— — flukes, 874
— — — varieties, 874
— — hepatic, 874
— — intestinal, 875

TROPICAL DISEASES, distomiasis, intestinal, etiology, 875
———flukes, 875, 876
————geographical distribution, 875, 876
———parasites, 875
———prophylaxis, 876, 877
———treatment, 876
————eucalyptus oil, 876
————male fern, 876
————thymol, 876
———trematodes, 875
————varieties, 875, 876
——treatment, 874
———cholecystotomy, 875
———prophylactic, 875
———salol, 874
—endemic hemoptysis, 877
——etiology, 877
——geographical distribution, 877
——parasites causing, 877
——prophylaxis, 877
——symptoms, 877
—filariasis, 880
——chyluria, 882
———arsphenamin in, 882
——Dracunculus medinensis, 880
———infection, 880
————mode, 880
———treatment, 882
————surgical, 882, 883
——Filaria bancrofti, 880
———mosquitoes and, 880, 881
———prophylaxis, 880
———treatment, 881
————ataxyl, 881
——Filaria loa, 883
———Calabar swellings, 883
———infection, 883
————mode, 883
———treatment, 883
————surgical, 884
——Filaria perstans, 884
——guinea worm, 882
——hematochyluria, 881
———treatment, 881
————quinin, 881
————x-ray, 881
——medina worm, 880
——parasites causing, 880, 882-884
——treatment, 881
———arsphenamin, 881
———surgical, 882
——worm of Pharoah, 882
—flagellate infections, 866
——Balantidium coli, 866
——ulcerative colitis, 866
———pathology, 866
———prognosis, 866
———symptoms, 866
———treatment, 867
————quinin enemata, 867
—frambesia tropica, 839
——diagnosis, 839
———differential, 839
————syphilis, 839, 840
——etiology, 839, 840

TROPICAL DISEASES, frambesia tropica, geographical distribution, 840
——pathology, 839, 840
——prophylaxis, 842
——symptoms, 841
——treatment, 841
———arsphenamin, 841
——Wassermann reaction, 839
—infections of bacterial or unknown etiology, 812
——dengue and denguelike fevers, 814
———cause, 815
————mosquitoes, Culex fatigans, 816
—————stegomyia, 816
———polymorphic character, 815
———prophylaxis, 816
———symptoms, 815
———treatment, 816
————symptomatic, 816
—————calomel purge, 817
—————fluid diet, 817
——hill diarrhea, 823
———etiology, 823
———symptoms, 823
———treatment, 823
————milk diet, 823
————saline laxative, 823
——local inflammations, 830
———climatic buboes, 833
————diagnosis, 833
————treatment, 833
———tropical ulcer, 832
————symptoms, 832
————treatment, 832
————dressings, protective, 832
———ulcerating granuloma of pudenda, 830
————causes, 831
————symptoms, 830
————treatment, 831
—————tartar emetic, 831
—————x-ray, 831
——low-country morning diarrhea, 823
———definition, 823
——Oroya fever, 828
———definition, 828
———prophylaxis, 828
————personal, 829
———symptoms, 828
———treatment, 830
————symptomatic, 830
—————blood transfusion, 830
—————drugs, 830
—————diet, light, nourishing, 830
—————rest, 830
——pappataci fever, 817
———definition, 817
———prophylaxis of, 818
———symptoms, 817
———treatment, 817
————symptomatic, 817
——references, 833-835
——sprue, 819
———confounded with pallagra, 820
———definition, 819
———etiology, 820
———pathology, 820

TROPICAL DISEASES, infections, sprue, prognosis, 821
——— symptoms, 820, 821
——— theories concerning, 819
——— treatment, 821
———— preventive, 821
————— climate, 821
———— specific, 822
————— vaccines, 822
———— symptomatic, 821
————— alcohol, interdiction of, 822
————— diet, 821
————— drugs, 822
————— milk diet, 822
————— rest, 821
————— saline aperient, 821
—— tsutsugamushi fever, 818
——— causes, 818
——— prophylaxis, 818
——— symptoms, 818
——— treatment, 819
—— undulant fever, 812
——— cause, 112
———— goat's milk, infected, 812
——— diagnosis, 813
———— agglutination tests, 813
——— symptoms, 812
——— treatment, 813
———— serum, specific, 813
———— symptomatic, 814
———— vaccines, 813
—— Verruga peruviana, 824
——— age and, 825
——— climatic conditions, 825
——— distribution, 824
——— etiology, 826
——— malaria and, 827
——— nature, 824
——— Oroya fever and, 827
——— prophylaxis, 824, 826
———— theories of immunization, 827
——— race and, 825
——— sex and, 825
——— treatment, 827
———— theories concerning, 827
— leeches, 888
—— protection against, 889
—— removal, 888
——— surgical, 888
—— species of, 888
— leishmaniasis, 858
—— dermal, 862
——— etiology, 863
——— geographical distribution, 863
——— lesions, 862, 863
——— prophylaxis, 864
——— treatment, 864
———— antimony, 864, 865
————— intravenous injection, 865
———— tartar emetic, 865
———— x-rays, 865, 866
—— references, 869
—— visceral, 858
——— complications, 861
———— treatment, 861, 862
——— etiology, 859
——— prophylaxis, 859

TROPICAL DISEASES, leishmaniasis, visceral, prophylaxis, insects and, 859
——— symptoms, 859
——— treatment, 858
———— antimony, 860
———— general, 862
———— sodium antimonyl tartrate, 861
———— tartar emetic, 860
————— complications following, 860
————— dosage, 860
————— intravenous injection, 860
——— types, 858
— nematode infection, 870, 884
—— Gnatostoma siamense, 888
—— Œsophagostomiasis, 887
——— parasites causing, 887
——— treatment, 887
———— thymol, 888
—— strongylosis, 886
——— geographical distribution, 886, 887
——— parasites causing, 886, 887
——— treatment, 887
———— vermifuge, 887
—— references, 889
—— Strongyloides stercoralis, 884
——— prophylaxis, 885
——— symptoms, 885
——— treatment, 885
—— trichocephaliasis, 886
——— prophylaxis, 886
——— treatment, 886
——— trichuris trichiura, 886
——— whip-worm, 886
— relapsing fevers, 836
—— diagnosis, 837
——— differential, 837
—— endemic, 836
—— Spirochæta recurrentis, 836
—— prophylaxis, 837
——— louse destruction, 837
——— tick destruction, 838
—— references, 867, 868
—— Spirochæta recurrentis, 836
——— strains, 837
—— symptoms, 837
—— transmission, 836
——— louse, 836
——— tick, 836
—— treatment, 838
——— medical, 838
——— symptomatic, 838
— schistosomiasis, 877
—— endemic hematuria, 877
——— geographical distribution, 877
——— parasites causing, 877, 878
——— pathology, 878
——— prophylaxis, 879
——— treatment, 878
———— hyoscyamus, 879
———— silver nitrate irrigations, 879
———— surgical, 879
———— urotropin, 879
—— intestinal, 879
——— geographical distribution, 879
——— parasite causing, 879
——— prophylaxis, 880

TROPICAL DIISEASES, schistosomiasis, intestinal, treatment, 879
———— sodium hypochlorid enemata, 879
———— quinin enemata, 879
—— Katayama disease, 880
——— geographical distribution, 880
——— parasite causing, 880
——— prophylaxis, 880
——— symptoms, 880
—— types of, 877
— spirochetoses, 836
— teniasis, 870
—— etiology, 870
——— Sparganum baxteri, 870
——— Sparganum mansoni, 870
——— Sparganum proliferum, 870
—— geographical distribution, 870
—— intestinal, 871
——— etiology, 871
——— parasites, 871, 872
——— prophylaxis, 871, 872
——— treatment, 873
———— male fern, 873
————— dosage, 873
—— prophylaxis, 871
—— somatic, 870
—— treatment, 871
——— surgical, 871
—— types of, 870
— theories concerning, 799-803
—— pigment in skin, relation of climate to, 801
—— thermoregulatory mechanism, relation of climate to, 801
—— tropical sunlight, injurious effects, 801
— trematode infection, 870, 875-877
— trypanosomiasis, 848
—— Chagas' disease, 856
——— diagnosis, 857
——— etiology, 856
——— prophylaxis, 857
——— symptoms, 857
——— transmission, 857
——— treatment, 857
—— forms of, 848
—— geographical distribution, 848
—— pathology of, 849
—— prophylaxis, 852
—— references, 868, 869
——— summary, 856
—— symptoms, 849
—— transmission of, 848
——— Tsetse fly, 848, 855
—— treatment, 849
——— antimony, 850
——— ataxyl, 850
——— "Bayer 205," 852
——— neokharsivan, 851
———— intravenous injection, 851
——— prophylactic, 852-856
———— drugs, 855, 856
———— general, 855
———— Tsetsefuges, 855
——— tartar emetic, 850
———— intravenous injections, 851
TROPICAL DISEASES, trypanosomiasis, treatment, tryparsamid, 851
———— experimental results, 851
——— vaccine, 850
———— experimental results, 850
— yellow fever, 842
—— etiology, 843
—— geographical distribution, 842
—— mortality, 847
—— onset, 843
—— pathology, 843
—— symptoms, 843
—— specific prophylaxis, 845
——— isolation, 845
——— mosquitoes, 845
———— breeding places, 846
————— destruction, 846
——— vaccination, 846
———— dosage, 847
————— duration, 847
———— immunity, 847
———— results, 847
—— transmission, 842
——— mosquitoes, 842
—— treatment, 844
——— general, 844
——— serum, 844
———— dosage, 845
———— results, 845
——— specific, 844
——— symptomatic, 844
TROPICAL INFECTIONS. *See* Tropical diseases.
TROPICAL LOCAL INFLAMMATIONS. *See* Tropical diseases, infections.
TROPICAL SUNLIGHT, effect on man of, 80
TROPICAL ULCER. *See* Tropical diseases.
TROTYL, as industrial poison. *See* Trinitrololuene.
Trypanocides, in trypanosomiasis, 851, 852
TRYPANOSOMIASIS. *See* Tropical diseases.
TSETSE FLY, in transmission of trypanosomiasis, 848, 854
Tsetsefuges, in prophylaxis of trypanosomiasis, 855
TSUTSUGAMUCHI FEVER. *See* Tropical diseases.
Tuberculin in, bronchial adenitis, 68
— renal tuberculosis, 613
— tuberculosis of the larynx, 27
— tuberculosis of mediastinal lymph nodes, 180
TUBERCULOSIS, CUTANEOUS. *See* Industrial diseases, dermatoses, occupational.
— in pneumocomosis, 773, 774
— of larynx. *See* Larynx, diseases of.
— of mediastinal lymph nodes. *See* Mediastinum, diseases of
TUBERCULOUS CYSTITIS. *See* Bladder, diseases of, cystitis.
TUBERCULOUS KIDNEY. *See* Kidney, diseases of.
TUBERCULOUS MYOSITIS. *See* Muscles, diseases of, 486
TUMOR, PELVIC, as a cause of Hydronephrosis, 592

TUMOR of, bladder. *See* Bladder, diseases of, 633
— lung. *See* Lungs, diseases of.
— mediastinum. *See* Mediastinum, diseases of.
— pleura. *See* Pleura, diseases of.
TURPENTINE, as industrial poison, 686

ULCER, TROPICAL. *See* Tropical diseases.
ULCERATING GRANULOMA OF PUDENDA, 830
ULTRAVIOLET RAYS in, insulation, 806
— occupational diseases, 721
UNDULANT FEVER. *See* Tropical diseases.
Unger method of transfusion, 215
UREMIA, 423
— chronic bronchitis and, 53
— hydronephrosis and, 595
— renal colic and, 602
UREMIA. *See also* Kidney, diseases of, hydronephrosis.
Ureteral transplantation, in bladder tumor, 649
Ureteral catheterism. *See* Catheterism.
Ureterolithotomy, in renal calculus, 607
Urethroscopic method of radiation, in bladder tumor, 643
Urinalysis, frequent, in phlebitis, 464
URINE, SUPPRESSION, in renal colic, 602
Urotropin in, enuresis, 626
— endemic hematuria, 879
— pyelitis, 585
Uterus, emptying of, in kidney of pregnancy, 578

Vaccination, prophylactic in, subacute bacterial endocarditis, 219
— — yellow fever, 846, 847
Vaccinations, desensitizing, in pulmonary emphysema, 117
Vaccines, antogenous, in chronic pneumonia, 112
Vaccines, streptococcic, in bronchopneumonia, 105
Vaccines in, acute rhinitis, 5
— chronic bronchitis, 53
— endocarditis, 212, 221
— enuresis, 628
— ozena, 19
— pyelitis, 588
— rhinoscleroma, 10
— sprue, 823
— tuberculosis of larynx, 27
— undulant fever, 813
VAGASTHENIA. *See* Irritable heart.
VALVULAR HEART DISEASE WITH CARDIAC PAIN, 291
Van Slyke's method of acidosis determination, 422
VASCULAR ACCIDENTS, in arteriosclerosis, 412
— *See also* Heart, diseases of.
Vegetable cathartics, in nephritis, 529, 553
Venesection in, anilin poisoning, 675
— anuresis, 504
— chronic myocarditis, 256
— congenital cardiac disease, 342
Venesection in, congestion of lungs, 92, 94
— kidney of pregnancy, 577
— large pulmonary emboli, 99
— methyl alcohol poisoning, 681
— nephritis, 542, 564
— pericarditis, 194
— pulmonary edema, 97
— thoracic aneurysm, 444
— uremia, 427, 428, 429
VENOMOUS ARTHROPODS. *See* Arthropods, venomous.
Ventilation, in prevention of caisson-disease, 699
Vermifuges in, intestinal distomiasis, 876
— strongylosis, 887
Veronal, in bronchitis, 43
VERRUGA PERUVIANA. *See* Tropical diseases.
VERTEBRATES, POISONOUS. *See* Poisons—reptiles—fish—arthropods and plants, affections due to.
VERTIGO, in caisson-disease, 694
VIOLET RAYS, effect of, on insolation, 806
VISCERAL LEISHMANIASIS. *See* Leishmaniasis.
VOMITING in, renal colic, 602
— yellow fever, 843

Waldenburg's pneumatic cabinet, in pulmonary emphysema, 118
WASPS, stings of, 896
WASSERMANN REACTION in, frambesia tropica, 839
— paroxysmal hemoglobinuria, 507
— syphilis of upper respiratory tract, 30
Water therapy in, anuresis, 503
— kidney of pregnancy, 577
— lead poisoning, 671
— phlebitis, 465
— pyelitis, 583, 585
— suppression of urine, 603
Whey cure, in chronic bronchitis, 52
WHIP-WORM, 886
Wiring and electrolysis, in thoracic aneurysm, 444
WORM OF PHARAOH. *See* Tropical diseases, filariasis
WRITER'S CRAMP, 778

"X" DISEASE, 313
Xenodiagnosis, in Chagas' disease, 857
XYLOL, as industrial poison. *See* Anilin.
X-ray, deep, and radium, in bladder tumor, 650
X-RAY BURNS. *See* Burns; X-ray dermatitis.
X-RAY DERMATITIS, 738
X-ray examination in, hydronephrosis, 594
— pneumocomiasis, 773
— renal calculus, 606
— renal tuberculosis, 610
X-ray therapy in, bladder tumor, 648
— bronchial adenitis, 68
— chronic bronchitis, 59
— chronic myocarditis, 229

X-ray therapy in, cutaneous tuberculosis, 796
— dermal leishmaniasis, 865
— eczema, 791
— epithelioma, 791
— hematochyluria, 881
— mediastinal tumors, 175
— occupational dermatoses, 792
— paronychia, 794
— tuberculous myositis, 486
X-ray therapy in, ulcerating granuloma of pudenda, 831
X-RAYS. *See* Occupational diseases, radiation.

YELLOW FEVER. *See* Tropical diseases.
YAWS. *See* Frambesia tropica.

ZAHN, EMBOLI OF. *See* Embolism.
ZINC, as industrial poison, 665
Zinc oxid, in industrial infections, 659

(1)

LIBRARY OF CONGRESS
0002612427A